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SPIDER-BITE IN SOUTH AFRICA *

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The existence of venomous spiders in Southern Africa has long been known but until the early part of the present century no definitely venomous species had been identified. In 1902 F. Pickard-Cambridge [1] described two species of *Latrodectus—L. indistinctus*, and *L. concinnus* which Smithers [9] in 1944 showed to be a synonym of *L. geometricus*.

From time to time cases of spider-bite were reported in the Union, particularly in the coastal belt of the Western Province. The cases varied in severity and in a number of instances serious illness and even death was recorded. These cases were attributed to a black spider locally known as the *knopiespinnekop*. In 1929 Dr. G. W. E. Macpherson [2] of Stellenbosch published a note on a series of 6 cases of spider-bite occurring in the Stellenbosch district. He stated that the cases were due to the bite of *Latrodectus maculata*. Specimens of this spider were later identified as *L. geometricus*. In 1936 and 1937 Finlayson [3, 4, 5] showed that the bite of the female of both *L. geometricus* and *L. indistinctus* could produce illness in man. The female of *L. indistinctus* was found to be the *knopiespinnekop* or Button spider.

Apart from the two species of *Latrodectus*, a member of the genus *Harpactirella*, *H. lightfooti* was reported by Finlayson and Smithers [6] in 1939 as producing two cases of arachnidism on Jutten Island, an island in Saldanha Bay, Cape.

DISTRIBUTION AND BIONOMICS

Smithers [9] (1944) investigated the distribution and bionomics of *L. indistinctus* and *L. geometricus*. He found that both species are widely spread throughout South Africa and the Rhodesias. Cases of spider-bite due to *L. indistinctus*, however, have only been reported from the wheat lands of the Western Province and of the Eastern Orange Free State. Illness due to the bite of *L. geometricus* has been reported from the Stellenbosch and Constantia districts.

Smithers found that *L. indistinctus*, amongst other sites, makes its nest in the wheat lands of the Western Province. During the harvest season these nests are disturbed and exposed to the sun, causing the spiders to seek shade and shelter in the sheaves of wheat which are left to dry in the fields. The workers collect several of these sheaves under their arms and may thus come into contact with a spider, which, being irritated by friction with the clothing or skin, may inflict a bite.

The majority of cases of spider-bite, none of which were fatal, caused by *L. geometricus*, have been reported as occurring in vineyards in the Western Province. This spider makes its nest in and around buildings, round barns, stables, garden buildings, under the bark of trees, under stones and in tufts of grass, bushes and vines. The reported cases have occurred in summer when grapes are being harvested and the spiders have been disturbed.

Four species of *Harpactirella*, an exclusively South African genus, have been described, all from the Cape Province. *H. lightfooti* was originally described from Paarl, but is apparently of much wider distribution than was at first supposed. These spiders are very active and aggressive and as they are hunting spiders they readily come into contact with man. The nests consist of silk-lined tunnels under stones, logs or other debris. *H. lightfooti* is a large spider with formidable fangs and closely resembles the non-venomous *Harpactira baviaana* locally known as the *Bobbejaanspinnekop*.

TOXICOLOGY

The venom of the South African species of *Latrodectus* was investigated by Finlayson [3, 4, 5] (1936, 1937), who showed that extracts of the cephalothoraces of these spiders contained a potent venom. The venom of the female *L. indistinctus* was, however, much more potent

* Abridgment of paper read before the First International Conference on Animal Venoms, 121st Annual meeting of the American Association for Advancement of Science, Berkeley, California, December 1954.

than that of the female *L. geometricus*, the minimum fatal dose of the venom of the former for rabbits being 3 mg. as against 12 mg. of the venom of the latter.

Cross neutralization and precipitin tests with the venom of *L. indistinctus* and *L. geometricus*, and antisera prepared with these venoms, showed that *L. indistinctus* venom not only contains the same antigens as *L. geometricus* venom but also an additional antigen. Antisera prepared against *L. indistinctus* venom could therefore be used in the treatment of spider-bite caused by either *L. indistinctus* or *L. geometricus*.

In 1945 Finlayson and Hollow [8] showed that a sample of purified *L. mactans* antiserum concentrate obtained from Dr. R. Sampayo was very effective in neutralizing the venom of *L. indistinctus* and in protecting mice against the bite of this spider. It would appear therefore that *L. mactans* and *L. indistinctus* venoms contain a common antigen and unless the venom of *L. mactans* contains additional antigens, the antigenic composition of the two venoms is identical, as complete protection against the bite of *L. indistinctus* was achieved by the injection of anti-mactans serum. Until the action of *L. indistinctus* antiserum on *L. mactans* venom is investigated the complete identity of the two venoms cannot be determined.

Whilst it was possible to demonstrate that *H. lightfooti* possessed a potent venom by causing the spider to bite mice and guinea pigs, the extreme lability of the venom prevented its isolation. It was therefore not possible for an antiserum to be prepared against this venom. Experiments with mice passively immunized with *L. indistinctus* antivenine and then bitten by *H. lightfooti* suggested that *L. indistinctus* antivenine conferred some protection, in experimental animals, against the bite of *H. lightfooti*.

TREATMENT OF SPIDER-BITE IN SOUTH AFRICA

Following the preparation of a potent antiserum against *L. indistinctus* venom, which was also shown to be effective against the venom of *L. geometricus*, this serum was issued on request to magistrates and district surgeons throughout the Union. In 1937 Finlayson [6] published a report on the effect of the antiserum in the treatment of 18 cases of spider-bite, all of which appeared to benefit from the serum injections. From time to time further reports on the efficiency of the serum in the treatment of 'Knopie-spider' bite were received, and it is of interest to note that no reports have been received of death in cases of spider-bite treated with serum, whereas before the issue of the serum a number of fatal cases of spider-bite had been reported in the Union.

During the period 1936-1945, 762 phials of antivenine were issued from the Union Health Department Laboratories at Cape Town and, from 1949 to 1953, 1,794 ampoules were issued by the South African Institute for Medical Research, Johannesburg, which undertook the manufacture and issue of the serum in 1949. It is evident from these figures that there has been a marked increase in the demand for *L. indistinctus* antivenine over the 4-year period 1949-1953 as compared with the 7-year period 1939-1945. No reports have been received of the effect of the serum in treating cases of bite by *H. lightfooti*.

Bogen,[10] discussing in 1955 the treatment of poisoning from the bite of *L. mactans* (Black Widow spider) in the U.S.A. stated that a multitude of therapeutic measures, including the use of convalescent serum and immune animal serum had been enthusiastically acclaimed and just as strongly flouted. He emphasized the need for controlled studies and critical consideration of the action of the various therapeutic measures and stressed the necessity for investigation of the treatments used.

It is not unreasonable to assume that the increased demand for *L. indistinctus* antivenine in the Union, results from its therapeutic efficiency. In view, however, of the paucity of reports on its efficacy since 1937, it is suggested that an estimation of the value of serum therapy in the treatment of spider-bite in South Africa should now be made.

REFERENCES

1. Pickard-Cambridge, F. (1902): Ann. Mag. Nat. Hist. (7) X, p. 38.
2. Macpherson, G. W. E. (1928): S. Afr. Med. J., **3**, 691.
3. Finlayson, M. H. (1936): *Ibid.*, **10**, 43.
4. Idem (1936): *Ibid.*, **10**, 735.
5. Idem (1937): *Ibid.*, **11**, 163.
6. Finlayson, M. H. and Smithers, R. (1939): *Ibid.*, **13**, 808.
7. Finlayson, M. H. (1955): Arachnidism in S. Africa, Trans. 1st Int. Conf. Animal Venoms. In press.
8. Finlayson, M. H. and Hollow, K. (1945): S. Afr. Med. J., **19**, 431.
9. Smithers, R. (1944): Ann. S. Afr. Mus., **36**, 263.
10. Bogen, E. (1955): *Treatment of Spider-bite Poisoning*, Trans. 1st Int. Conf. Animal Venoms. In press.

BOOKS RECEIVED : BOEKE ONTVANG

The Year Book of Neurology, Psychiatry and Neurosurgery (1954-1955 *Year Book Series*). Edited by Roland P. Mackay, M.D., S. Bernard Wortis, M.D., Percival Bailey, M.A. and Oscar Surgar, M.D. Pp. 619 with 97 illustrations. $7.00. Chicago: Year Book Publishers, Inc. 1955.

Ciba Foundation Colloquia on Endocrinology. Volume VIII The Human Adrenal Cortex. Edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., Margaret P. Cameron, M.A., A.B.L.S. and Joan Etherington. Pp. 665+xv with 227 illustrations. 55s. London: J. &. A. Churchill, Ltd. 1955.

Storkie's Footsteps, captions by Oom Harry and lino-cuts by Joan. Pp. 53 with illustrations. 7s. 6d. Obtainable at C.N.A.

Ooievaarspore, woorde deur Oom Harry en linosnitte deur Joan. 53 bladsye met illustrasies. 7s. 6d. Verkrygbaar by die Sentrale Nuusagentskap.

A Survey of the Food and Feed Resources of the Union of South Africa. By G. van de Wall, M.Sc. Agric. (Pret.) and E. D. Alvord, Jnr., M.Sc. Agric. (Pret.). Pp. 312+xiv, with figures. Pretoria: J. L. van Schaik, Ltd. 1954.

Basic Medical Terms and Techniques Simplified. By Minnie I. Paddock. Pp. 148+v. Chicago: American Technical Society. 1955.

Stammering: Its Cause and Cure. A Supplement to Stammer is not Nerves. By H. V. Hemery, L.R.A.M. Pp. 17+iv with 5 illustrations. London: The School for Functional Speech Disability. 1955.

The Year Book of Orthopedics and Traumatic Surgery. (1954-1955 *Year Book Series*). Edited by Edward L. Compere, M.D., F.A.C.S., F.I.C.S. Pp. 384+193 illustrations. Chicago: Year Book Publishers, Inc. 1955.

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VAN DIE REDAKSIE

LIGGING VAN DIE LETSEL IN LONGTERING

Dit was Valsalva wat die aandag daarop gevestig het dat die letsels van longtering dikwels tot die boonste lobbe beperk is, en Morgagni het 'n stap verder gegaan deur aan te teken dat hulle die boonste dele van die lobbe verkies. Laennec het opgemerk dat hulle nie net die bopunte aanval nie maar dat die regterkant meer dikwels aangetas word as die linkerkant. Maar onlangs nog kon Rich konstateer dat dit nog nie moontlik is om die lokalisasie aan die bopunt bevredigend te verklaar nie.

In 1887 het Orth gesuggereer dat daar 'n verband bestaan tussen hierdie lokalisasie en anemie van die longpunt met verminderde limfvorming. Hy het die hoë voorkomssyfer van longtering by pasiënte met stenose van die longslagaarklep aangehaal as 'n argument ten gunste van hierdie teorie. Bloomfield *et al*[1] het hierdie teorie onderskryf toe hulle opgemerk het dat die longslagaardruk te laag was in vergelyking met die swaartekrag om die bloedvloei in die boonste derde van die longe te handhaaf wanneer 'n volwassene sit of staan. Die mening was dat die gevolglike verandering in gasdruk in die aangetaste dele die groei van teringkieme bevorder, maar wat as van groter belang beskou was is die verminderde limfvloei en die staking van toksienverdunning deur plasmafiltraat as gevolg van die verminderde bloedvloei. Dock[2] het dit benadruk dat die waarde van rus in die bed by die behandeling te danke is aan die herstel van die normale vloei in die bodele van die longe.

Die posisie van die liggaam as 'n faktor in die lokalisasie van die siekte, moontlik as gevolg van veranderinge in gasdruk, word in die laaste tyd benadruk.

Aldus het Medlar[3], in proefnemings met beeste en konyne, opgemerk dat die letsels in kroniese gevalle in die juxtafreniese deel naby die dorsale oppervlakte van die longe te vinde was. Deur konyne met die kop en skouers na bo te laat hang vir periodes van 11 uur per dag het die letsels hoofsaaklik in die punte voorgekom soos in die mens. Ander het daarop gewys dat in die vlermuis, wat met sy kop na ondertoe slaap, die grootste knoppies naby die middelrif gevind word.

Scott *et al*[4, 5, 6] dink nie dat die verminderde limfvloei van groot belang is nie. Hulle stel voor dat veranderinge in gasdruk wat deur verandering van bloedvloei in die longe geskep word van die allergrootste belang is vir die bevordering van die groei van teringkieme. Hul anastomose-eksperimente het bewys dat ruime volume en hoë druk van die bloed wat die long binnekom geen teëhoudende effek op teringknoppies het nie, so lank as geen deel van die onversadigde bloed wat deur die are

EDITORIAL

SITE OF THE LESION IN PULMONARY TUBERCULOSIS

It was Valsalva who pointed out that the lesions of pulmonary tuberculosis are often confined to the upper lobes, and Morgagni went a stage further by noting that the lesions favoured the upper part of the lobes. Laennec noted not only that they attacked the apex, but that the right apex was more commonly affected than the left. But only recently Rich was able to state that it is still not possible to explain satisfactorily the apical localization of the disease.

Orth in 1887 had suggested that this localization was related to an apical anaemia with reduced lymph formation. He instanced the high incidence of pulmonary tuberculosis in patients with stenosis of the pulmonary valve as a point in favour of this theory. Bloomfield *et al.*[1] supported this theory when they noted that pulmonary arterial pressure was too low in comparison with the force of gravity to maintain the flow in the upper third of the lungs when adults were sitting or standing. The resulting change in gas tension in the affected zones was believed to favour growth of tubercle bacilli, but what was thought to be more important was the reduction of lymph flow and cessation of the dilution of toxins by plasma filtrate as the result of the diminished blood flow. Dock[2] stressed that the value of bed rest in treatment was due to restoration of the normal flow in the upper part of the lungs.

Posture as a factor in the localization of the disease, possibly as a result of changes in gas tension, has been stressed lately.

Thus Medlar,[3] working on cattle and rabbits, noted that the lesions in chronic cases were to be found in the juxta-phrenic region, near the dorsal surface of the lungs. By holding rabbits suspended with the head and shoulders uppermost for periods of 11 hours per day the lesions occurred mainly in the apices, as in man. In the bat, which sleeps with head hanging down, others have shown that the largest tubercles are found near the diaphragm.

The importance of reduction of lymph flow has been minimized by Scott *et al.*[4, 5, 6] who suggest that changes in gas tension created by altered pulmonary flow are of paramount importance in favouring the growth of tubercle bacilli. Their anastomosis experiments have shown that ample volume and high pressure of arterial

van die liggaam na die hart terugvloei die lugsakkies van die long bereik nie. Blykbaar is dit teenstrydig met die teorie dat verminderde bloedvloei met die verminderde limfvloei en verminderde toksienverdunning wat daaraan verbonde is die belangrike faktor in die lokalisasie van die letsel is.

Ook Rich en Follis[7] meen dat gasdruk van groot belang kan word. Hulle het getoon dat teringletsels in marmotjies gestuit kan word deur die diertjies lug met so min as 10% suurstof te laat inasem; en dit ten spyte van die oormatige lugtoevoer wat deur suurstofgebrek en die vermeerderde werk van die longe veroorsaak word.

Dit is bevind dat hidrodinamiese faktore die feit kan verklaar dat aanvangsletsels 50% meer dikwels in die regter bolob as in die linker een voorkom. Die hooflongslagaar, die linkerlongslagaar en die slagaar na die linker bolob loop in 'n meer reguit lyn dan wat die geval is aan die regterkant, waar die regter longslagaar met 'n reghoek wegdraai by die verdeling en die tak na die bolob weer met 'n skerp hoek wegdraai by sy oorsprong. Dus sal die drukking in die regter bolob laer wees dan in die linker een, en die streek van die long met hoë suurstof- en lae koolsuurgasdruk sal dieper aan die regterkant as aan die linkerkant strek.

Die teenswoordige werk wil aandui dat liggings-, hidrostatiese en gasdruk-verskille die lokalisasie van die letsels in die longe bepaal. Die doeltreffendheid van longkollaps as behandeling is moontlik te danke aan die vermindering van bloedvloei en veranderinge in longsuurstof en -koolsuurgas tot die peil van dié in die are. Die hoë voorkomssyfer van longtering in stenose van die longslagaarklep, dielae voorkomssyfer daarvan in myterklepstenose en die doeltreffendheid van platlê in die bed vir die stuiting en herstel van vroeë longpunttering kan moontlik almal deur hierdie faktore verklaar word.

In 'n onlangse referaat het Dock[8] die interessante werk wat gedoen is volledig opgesom. Soos hy aandui het die ontwikkeling van antibiotikas die waarde van rus in die bed vir lang tydperke heelwat gewysig. Mens sou daarby kon voeg dat die stryd teen ondervoeding, oorbevolking en agterbuurtes moet voortgaan as tering uitgeroei moet word.

1. Bloomfield, R. A., Lauson, H. D., Cournand, A., Breed, E. S. en Richards, D. W. Jr. (1946): J. Clin. Invest., **25**, 639.
2. Dock, W. (1946): Amer. Rev. Tuberc., **53**, 293.
3. Medlar, E. M. en Sasano, K. T. (1936): *Ibid.*, **34**, 456.
4. Scott, H. W. Jr., Hanlon, C. R. en Olson, B. J. (1950): J. Thorac. Surg., **20**, 761.
5. Hanlon, C. R., Scott, H. W. Jr. en Olson, B. J. (1950): Surgery, **28**, 209.
6. Olson, B. J., Scott, H. W. Jr., Hanlon, C. R. en Mattern, C. F. T. (1952): Amer. Rev. Tuberc., **65**, 48.
7. Rich, A. R. en Follis, R. H. Jr. (1942): Trans. Assoc. Amer. Phys., **57**, 271.
8. Dock, W. (1954): Arch. Intern. Med., **94**, 700.

blood entering a lung have no retarding effect on tubercles as long as none of the unsaturated blood returned to the heart by the systemic veins reaches the alveoli. This would appear to controvert the theory that diminished blood flow with associated lessened lymph flow and lessened dilution of toxins is the important factor in the localization of the lesion.

That gas tension may be of great importance is also suggested by Rich and Follis,[7] who showed that tuberculous lesions in guinea-pigs can be arrested by making the animals breathe an atmosphere containing as little as 10% oxygen; and this in spite of the hyperventilation caused by the anoxia and the increased work of the lungs. Hydrodynamic factors have been found to explain the fact that the incidence of initial lesions is 50% greater in the right upper lobe than in the left. The main pulmonary artery, the left pulmonary artery and the artery to the left upper lobe are in a straighter line than is the case on the right, where the right pulmonary artery turns at an acute angle at the bifurcation, and the branch to the upper lobe turns at an acute angle at its origin. Thus the pressure head will be lower in the right upper lobe than in the left, and the zone of lung with high oxygen and low carbon-dioxide pressures will extend more deeply on the right side than on the left.

The present work tends to indicate that postural, hydrostatic and gas-tension differences determine localization of lesions in the lungs. Possibly lung collapse owes its efficacy in treatment to the reduction in blood flow and changes of pulmonary O_2 and CO_2 to the levels in venous blood. The high incidence of pulmonary tuberculosis in pulmonary-valve stenosis, its low incidence in mitral stenosis and the effectiveness of lying flat in bed for the arrest and healing of early apical tuberculosis may all be explained by these factors.

Dock,[8] in a recent paper, fully summarizes the interesting work which has been done. As he points out, in treatment the value of bed rest for long periods of time has been greatly modified by the development of antibiotics. One would add that the fight against malnutrition, overcrowding and slum conditions must go on if tuberculosis is to be eradicated.

1. Bloomfield, R. A., Lauson, H. D., Cournand, A., Breed, E. S. and Richards, D. W. Jr. (1946): J. Clin. Invest., **25**, 639.
2. Dock, W. (1946): Amer. Rev. Tuberc., **53**, 293.
3. Medlar, E. M. and Sasano, K. T. (1936): *Ibid.*, **34**, 456.
4. Scott, H. W. Jr., Hanlon, C. R. and Olson, B. J. (1950): J. Thorac. Surg., **20**, 761.
5. Hanlon, C. R., Scott, H. W. Jr. and Olson, B. J. (1950): Surgery, **28**, 209.
6. Olson, B. J., Scott, H. W. Jr., Hanlon, C. R. and Mattern, C. F. T. (1952): Amer. Rev. Tuberc., **65**, 48.
7. Rich, A. R. and Follis, R. H. Jr. (1942): Trans. Assoc. Amer. Phys., **57**, 271.
8. Dock, W. (1954): Arch. Intern. Med., **94**, 700.

ADRENOCORTICAL HYPERFUNCTION

2. ANDROGENS AND MINERALOCORTICOIDS

In an editorial in the last issue of the *Journal* the features caused by an excessive production of glucocorticoid secretion, known as Cushing's syndrome, were considered. The second group of hormones formed by the adrenal cortex are androgens (the 'N' or 'nitrogen' substances of Albright, so-called because of their anabolic activity and hence their tendency to cause nitrogen retention).

The symptoms of the adreno-genital syndrome are

produced by excess of these masculinizing hormones, as is indicated by the finding of a high urinary 17-ketosteroid excretion. As in Cushing's syndrome there may be an adenoma, a carcinoma or bilateral hyperplasia of the cortex to account for the hypersecretion. Where, however, congenital adrenal hyperplasia is present it is considered not to be the basic defect, but rather that an isolated inability to synthesize glucocorticoids is the primary lesion. In these patients the circulating level of 17-hydroxycortosteroids is abnormally low, and there is little or no increase following stimulation with ACTH, whereas the excretion of 17-ketosteroids in the urine is much increased and is even further raised after ACTH administration. [2]

The syndrome may now be explained by invoking the idea of thermostat-like regulation of the pituitary. Normally the circulating glucocorticoids damp down the production of ACTH by the pituitary, which in turn diminishes the stimulation of the adrenal cortex and tends to lower glucocorticoid formation. Thus the body regulates its level of circulating cortical hormones. However, when the glucocorticoids are abnormally depressed, ACTH production is greatly enhanced and the other hormones produced by the adrenal cortex are secondarily increased, particularly the androgens. (The position of the mineralocorticoids is less clear, though in some cases of congenital adrenal hyperplasia they may be deficient, in which circumstances an Addisonian state occurs in combination with the adrenogenital syndrome.) Further confirmation of the above hypothesis is to be found in the effect of administered cortisone or hydrocortisone on the androgen excretion in this syndrome. Not only does this drop to normal levels (as indicated by the urinary 17-ketosteroids) but the patient's symptoms quite rapidly revert to normal in a remarkable manner.[3] On the other hand cortisone has no effect on the syndrome when it is caused by a tumour.

The so-called 'adreno-genital syndrome' really consists clinically of a group of 3 syndromes, one appearing in young girls, one in young boys and the third in older girls or in women. The condition of congenital adrenal hyperplasia in young girls is by far the commonest cause of female pseudo-hermaphroditism. The symptoms are variable and include evidence of masculinization such as an enlarged clitoris, persistent urogenital sinus, and early growth of pubic and axillary hair. Later it is evident that female secondary sex-characters are not appearing, male type of hair grows on face and body, and the voice deepens. Failure of normal salt retention may occur in infancy with death in Addisonian crisis. On the other hand some patients develop hypertension.

Adult females complain of secondary amenorrhoea and sterility and show varying degrees of virilization: growth of hair on face and body but recession at the temples, enlargement of clitoris and thyroid cartilage, deepening of voice, coarsening of features with acne, increase in muscularity. There are, of course, very many women who suffer from hirsutes with masculine distribution of hair (though not usually temporal recession) and who appear to have no underlying endocrine disturbance whatever. On the other hand Kinsell [4] has reported one case in which hirsutes was the sole clinical manifestation of an adrenal cortical tumour (though in the protocol remark is made of great muscular development). The 17-ketosteroid output in this case was between 60 and 90 mg. per day.

In boys the condition of 'macrogenitosomia praecox' is produced—'the pocket Hercules' type. The boys develop a 'pseudo-precocious puberty' with advanced bone age, muscular development, sexual hair growth, penile size and voice change. The testes may be much less enlarged and spermatogenesis is, naturally, not to be expected. As in the females, the 17-ketosteroids are very high, and an Addisonian state may be present in those cases which are due to congenital adrenal hyperplasia.

Finally it may be repeated that hyperplasia or carcinoma of the adrenal cortex may cause a mixed picture of Cushing's syndrome and the adreno-genital syndrome, in which both glucocorticoids and androgens are produced in excess.

The third group of hormones manufactured in the adrenal cortex are the mineralocorticoids, the 'salt-retainers', represented by aldosterone. Excessive production of these substances may possibly account for the hypertension in some of the cases of congenital adrenal hyperplasia. Conn [5] has recently described a condition of 'primary aldosteronism,' in which patients develop intermittent tetany with normal serum calcium, paraesthesiae, periodic severe muscular weakness, polyuria and polydipsia, hypertension without oedema. Biochemically there is hypokalaemia, hypernatraemia, alkalosis and an inability of the renal tubules to reabsorb water. Excessive amounts of a salt-retaining corticoid are found in the urine.

The field is wide open for workers to ascertain what part, if any, aldosterone excess plays in essential hypertension, in the oedema of nephrosis and cirrhosis of the liver, in cardiac failure and in states of 'stress' such as attends a major operation. It is becoming more and more evident that endocrinology will play a fundamental part in explaining the basic features of many syndromes apparently belonging to other specialities.

REFERENCES

1. S. Afr. Med. J. (1955): **29.**
2. Bongiovanni, A. M., Eberlein, W. R. and Cara, J. (1954): J. Clin. Endocr., **14,** 409.
3. Wilkins, L., Lewis, R. A. and Klein, R. (1950): Bull, Johns Hop. Hosp., **86,** 249.
4. Kinsell, L. W. and Lisser, H. (1952): J. Clin. Endocr., **12,** 50.
5. Conn, J. (1954): Brit. Med. J., **2,** 1415.

RIFT VALLEY FEVER IN SOUTH AFRICA

A STUDY OF THE 1953 OUTBREAK IN THE ORANGE FREE STATE, WITH SPECIAL REFERENCE TO THE VECTORS AND POSSIBLE RESERVOIR HOSTS

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The occurrence of Rift Valley fever in South Africa was first recognized in 1951.[1] In the autumn of that year an extensive and severe epizootic occurred in the Western Free State, the Southern and South-Western Transvaal, and the adjoining districts of the North-Western Cape Province.[2, 3] Many farmers lost nearly all their lambs and a large proportion of their sheep. The loss of cattle was less severe. At the same time as the occurrence of this epizootic amongst sheep and cattle there were a number of cases of illness amongst the farmers and their farm labourers. Several veterinary surgeons who had done post-mortem examinations on infected sheep or cattle also developed a similar illness. All these patients had cut open or had handled the viscera and meat of sheep and cattle. It was clear that only those individuals who handled infected tissues contracted the disease, which was proved by the isolation of virus and serological tests to be Rift Valley fever. The infection did not spread from patients to individuals coming into contact with them. Several patients developed visual defects in convalescence. This defect was found to be due to a circumscribed retinitis.[4, 5] In some, after may months, the lesions have resolved; in others the defect still persists 3 years later.

Laboratory studies, carried out towards the end of the epidemic, failed to reveal the vector amongst the mosquitos frequenting and breeding in the pans, and also failed to reveal infection amongst the few wild animals examined.

There seemed little doubt that the disease was recently introduced into this region. The oldest farmers in the affected districts recognized it as a new disease, a view endorsed by veterinary officers of many years' experience. Their opinion that it was a new and hitherto unknown infection in this region was strongly supported when, in spite of their many years' experience and their long years' residence in contact with sheep and cattle, they became ill soon after their contact with the tissues of sheep and cattle. If they had had previous contact with the infection they would have been immune. Where it came from remains uncertain. Whether it would persist in this region of South Africa was also a most important question, which has now been partly answered.

1953 OUTBREAK

In the autumn of last year, 1953, the disease once again appeared in epizootic form in the Luckhoff District of the Orange Free State. This district was not affected in 1951, but adjoins the Koffiefontein District, which was involved in the first epidemic. Within a month several hundred sheep died after an acute short illness characterized by stiff gait, weakness, and sometimes bleeding from the nose and intestine. The illness was so acute that often sheep were found dead in the morning, when the previous evening they had not appeared to be ill. Cattle were not obviously affected.

Several of the farm labourers on the affected farms were ill at the same time as the epizootic occurred amongst the sheep. The individuals affected had cut open dead sheep or handled their meat. A Native woman working in the kitchen where she handled meat from slaughtered sheep was also affected.

Most of these patients had a diphasic illness, with fever lasting 3 days, followed by one day's remission, followed by a further 3 days' fever. During the illness the patients suffered from severe headache, backache, and muscular pains. One of the patients complained in convalescence that his head still did not feel right and that he could no longer see clearly, being unable to identify objects more than 25 yards away. It will be recalled that a diphasic illness followed in some cases by defective vision was a feature of the human cases in the 1951 outbreak of Rift Valley fever. This diagnosis in the present outbreak was confirmed retrospectively by finding that the sera from these patients gave a positive mouse-protection test, as will be described later.

The nature of the new epidemic was immediately suspected by Dr. van der Linde and Dr. Dickson of the Department of Veterinary Services. In consultation with Dr. R. A. Alexander, the Director of Veterinary Services, arrangements were made to send a team from the South African Institute for Medical Research and the Medical Ecology Laboratory of the Union Health Department to study the outbreak in the field. A camp was established on Mr. P. Gouws' farm 'Legpan'. The study included the situation on this farm and the neighbouring farms 'Gannapan', 'Eldorado', and 'Wolveplaat'. These farms are about 15 miles north of Luckhoff in the South-West Free State. The veld in this area is covered by Karroo bush. There are several large pans which fill up in the rainy season, but the water soon evaporates in the dry season and leaves a dry caked and cracked surface. Gannapan, the largest of these pans, covers about 600 morgen (1,300 acres) and has a very rich and varied bird life, including spurwing geese, Egyptian geese, various species of wild duck, stilts, plover, and blue cranes, as well as large numbers of the smaller species. There are

also blesbok, springbok and steenbok on the lands adjoining the pan.

These pans form the breeding places of several species of mosquitos, particularly *Aedes caballus*, *Culex theileri* and *Anopheles squamosus*, and at dusk large numbers of these mosquitos rose to feed. It was noted that they fed actively on sheep, cattle, man and experimental mice. The sheep in the lands immediately adjoining the pans were most severely affected in the outbreak.

ISOLATION OF VIRUS FROM SHEEP

A sheep found recently dead on the veld was opened, with a sterile syringe and needles about 10 c.c. of blood was removed from the heart, and a piece of liver was excised and placed in a sterile bottle.

On return to the camp, a suspension of the liver was prepared and inoculated intraperitoneally into 8 mice. These died on the 2nd and 3rd day after inoculation. Sections of the liver showed the total necrosis typical of Rift Valley fever. Virus-neutralization tests with antisera against known strains of Rift-Valley-fever virus confirmed that this virus was the virus of Rift Valley fever.

The serum was separated from the blood and submitted to a complement-fixation test for Rift Valley fever. This test gave a negative result, which is not surprising as the disease is so rapidly fatal that there is often no time for the formation of antibodies.

The blood clot was suspended in saline and the suspension inoculated into embryonated hen eggs, and a virus, later proved to be the virus of Rift Valley fever, was successfully established in egg culture.

VECTOR STUDIES

Isolation of Virus from Arthropods

Batches of mosquitos were collected by the team at various sites in the area. Most of these batches were caught in the late afternoon on Gannapan. They included *Aedes caballus*, *Culex theileri*, and *Anopheles squamosus*.

In the evening these batches were allowed to feed on mice in a mosquito cage. It was noted that about 50% of the mosquitos fed.

The following morning the fed mosquitos were separated from the unfed. Each batch was then killed by ether or chloroform vapour. A suspension of the mosquito bodies was prepared in saline and 0·1 c.c. of this suspension inoculated in 4–8, most often 5, mice. These mice were observed for up to 14 days, when they were challenged with known Rift-Valley-fever virus. The results of these tests are given in Table I.

In the table, the feeding experiments' number is followed by the letter A, and the relation of A batches to B batches is, for example, as follows: *Batch* 2A—Mice fed to mosquitos; *Batch* 2B—Mosquitos in collection A inoculated into mice.

It will be noted that a virus pathogenic for mice was isolated from 6 batches of *Aedes caballus* and from 3 batches of *Culex theileri*. It was also shown that the infection could be transmitted by the bite of *Aedes caballus* with naturally acquired infection.

TABLE I. VIRUS ISOLATION FROM INSECTS

Date (May 1953)	*Exp. No.*	*Insect Species*	*Route of Inoc. of Mice*	*Result*
10	1	Aedes caballus	IP	negative
,,	2	Aedes caballus	IP	positive
,,	5	Simulium sp.	IP	negative
,,	6	Aedes caballus	IP	positive
12	2 A	Aedes caballus	feeding	negative
13	2 B	Aedes caballus	IP	positive
,,	3 A	Aedes caballus and Culex theileri	feeding	negative
,,	3 B 1	Aedes caballus	IP	positive
,,	3 B 2	Culex theileri	IP	negative
,,	3 B 3	Aedes caballus	IP	positive
,,	3 B 4	Culex theileri	IP	positive
,,	4 A	Aedes caballus	feeding	positive
14	4 B 1	Aedes caballus	IP	positive
,,	4 B 2	Anopheles squamosus ..	IP	negative
,,	5	Stomoxys sp.	IP	negative
,,	9	Simulium sp.	IP	negative
,,	11	Culex theileri and Anopheles squamosus ..	feeding	negative
16	15	Culex theileri, fed	IP	negative
,,	16	Culex theileri, unfed ..	IP	negative
,,	17	Anopheles squamosus ..	IP	negative
,,	18	Culex sp. (theileri) ..	IP	positive

Summary: positive virus isolations—
7 from *Aedes caballus* 1 feeding.
3 from *Culex theileri*.

Identification of Virus—Pathological Findings

The mice dying from this infection were found to have an extensive, almost total, destruction of the liver. The parenchymal cells show an eosinophilic degeneration of the cytoplasm; often the eosinophilic material is rounded up to form an inclusion body somewhat resembling the Councilman body characteristic of yellow fever in man. The nucleus of the affected cells show margination and fragmentation of the chromatin and often small intranuclear eosinophilic inclusion bodies. In addition, fragments of pyknotic nuclei are scattered through the substance of the liver and may often be found within the Kupfer cells. Similar pyknotic fragments may be seen in cells of the spleen pulp and lymph glands.

Although degeneration is so extensive there is little cellular infiltration, but neutrophil leucocytes and mononuclear cells have increased, and often there is marked congestion, red cells filling and greatly distending the sinusoids of the spaces between the degenerate and often dissolved and disappearing columns of liver cells.

This pathological picture is characteristic of Rift Valley fever in mice and sheep. A somewhat similar picture is seen in liver sections of human beings who have died of yellow fever. However, the virus of yellow fever does not produce liver lesions in mice and, unlike the virus of Rift Valley fever, is ralatively innocuous to mice when inoculated intraperitoneally. No other virus is known to produce similar liver lesions.

Immunological Findings

The viruses isolated from the sheep and the batches of mosquitos were proved by cross-immunity tests to be

the Rift-Valley-fever virus. A group of monkeys were inoculated with suspensions of infected mouse liver obtained from the mice infected with the Luckhoff sheep strain, the *Aedes caballus* strain, and also with the Ben strain isolated in 1951, and with the Smithburn neurotropic strain isolated in Uganda.

A fortnight later these monkeys were bled by intracardiac puncture and 10-20 c.c. of blood taken. The sera separated from these bloods were tested for their neutralizing power against homologous strains and

TABLE II. RESULTS OF PROTECTION TESTS WITH ANTISERA

Result = Survivors/total

Monkey Antisera		*Smithburn Strain*	*Ben* 1951 *Strain*	*A. caballus* 4*A strain* 1953	*Sheep* 1953 *strain*
Smithburn	N	0/5			
	I	5/5			
Ben 1951	N	0/5	1/5	1/5	1/5
	I	3/5	5/5	4/5	4/5
A. caballus	N	0/5	0/5	1/5	2/5
4A—1953	I	3/4	4/5	3/4	5/5
A. caballus	N	1/5	0/5	2/5	0/5
4B—1953	I	5/5	5/5	5/5	5/5
C. theileri	N		0/5		
3B4—1953	I		5/5		
Sheep 1953	N	1/5	0/5	2/5	
	I	4/5	4/5	5/5	
6	N	1/5	0/5		
	I	4/4	5/5		
Human Immune Serum			5/5	5/5	5/5

against the Ben (1951) and Smithburn strains respectively. The results are given in Table II. This test was repeated with some additional antisera included, with the results shown in Table III.

It was shown that the sera from the Ben and Smithburn monkeys neutralized their homologous virus and also the strains isolated in the 1953 Luckhoff outbreak,

TABLE III. MOUSE-PROTECTION TESTS WITH PREPARED ANTISERA

Antisera	*Homologous Virus*	*Virus Challenge Ben S.A.* 1951	*4 B I S.A.* 1953
Ben 1951		5/5	5/5
Smithburn	5/5	5/5	5/5
A. caballus (6) 1953	5/5	4/4	3/3
A. caballus 4*B* 1953		5/5	5/5
Sheep S.A. 1953	5/5	5/5	5/5
C. theileri 3 *B*4 1953	4/4	4/4	5/5
Human sera		0/5	
negative 1		0/5	
2		0/5	
3		0/5	
positive 1		5/5	
2		5/5	

and *vice versa*. There was thus cross-immunity confirming the identity of these strains as strains of Rift-Valley-fever virus.

More detailed studies will be undertaken to determine whether there are any antigenic differences within the group, but these minor differences do not affect the main conclusions, which are that the outbreak in the Luckhoff District in 1953 was caused by Rift-Valley-fever virus, and that the infection was harboured by *Culex theileri* and *Aedes caballus*, and further that the latter mosquito can transmit the infection whilst feeding.

Search for Animal Reservoir Hosts

The presence of the virus in sheep and mosquitos having thus been shown, arrangements were made for a second expedition to collect blood from small mammals and birds, and large numbers of ectoparasites, with the object of discovering a possible reservoir among wild animals or birds and ectoparasites capable of harbouring the virus.

A systematic collection of the animals of the veld of the affected area was made by R. Rose Innes and K. H. Schulz of the Medical Ecology Laboratory of the Union Health Department, assisted by A. C. Pelzer of the staff of the Deputy Chief Health Officer of the Union in Bloemfontein. During a period of 12 days, 100 bloods were collected from 15 species of small mammals and 14 bloods from 6 species of birds. Liver specimens were taken from most of these animals and birds. In addition, 13 large batches of ectoparasites (fleas, mites and ticks) were collected from the pooled nests of 4 different species of rodents. Twenty-nine smaller batches of ectoparasites were collected from the bodies of small mammals, more than half of which were from specimens already sacrificed for blood. Finally, 25 cows (23 infested), 300 sheep (59 infested) and 4 horses (all infested) were searched and stripped of their ectoparasites.

All the ectoparasites were identified in the Department of Entomology of this Institute before being tested for the presence of virus.

The sera from the blood specimens were separated and the serum from each animal was submitted to a Rift-Valley-fever mouse-protection test.

Portions of liver from the various animals and birds were taken and placed in 5% formol saline. These were embedded and histological sections were examined microscopically for lesions of the liver, particularly lesions which may have resulted from a previous recent attack of Rift Valley fever.

Suspensions were prepared from each lot of arthropods by grinding them up with a pestle and mortar and adding normal saline. Each suspension was then inoculated into 5 mice, which were observed for signs of illness for 1 month.

At the end of this time the surviving mice were challenged with a known Rift-Valley-fever strain of virus to determine whether they had undergone an inapparent infection during the period of observation.

Mice which became seriously ill were killed and portions of their viscera, including the brain, liver, heart and lungs, spleen and kidney, were removed. These were placed in formalin or Bouin's fixative before embedding for sectioning for histological examination.

At the same time a portion of liver was removed aseptically and stored at —20° C, pending the result of the histological and bacteriological examination. If this suggested or confirmed the suspicion of Rift Valley fever, material was available for passage and further study.

The results of the tests for Rift-Valley-fever virus in the ectoparasites of the veld animals were all negative, as is shown in Table II.

Although many of the liver sections showed pathological lesions none of these resembled the lesions of Rift Valley fever.

The Rift-Valley-Fever Mouse-protection Test

The blood sera from 4 of the farm labourers who had been ill, as well as from the animals collected by the second expedition, were submitted to a Rift-Valley-fever mouse-protection test. In this test, 0.5 c.c. of serum, or less if no more was available, was mixed with an equal quantity of a virus suspension containing 50-1,000 mlds. per 0.1 c.c. The mixture was thoroughly shaken and then incubated at 37° C. for 1 hour, being shaken at 10-minute intervals during this time.

Then each serum-virus mixture was inoculated intraperitoneally in 0.1-c.c. amounts into each of 5 mice. These mice were observed for 7 days, a daily note being made each day of their state. The interpretation of the results was: if 4 or 5 survived = positive protection; if 2 or less survived = negative = no protection; if 3 survived = inconclusive.

The results are given in Table III. The 4 human sera were found to be protective, thus confirming the diagnosis made clinically of Rift Valley fever. The only animal giving a postive Rift-Valley-fever protection-test was a *muishond* or polecat (*Ictonyx sp.*). As this animal is known to eat dead lambs it is possible that it acquired its infection from direct contact with the infected tissues and not from an arthropod vector. Whether this animal plays any part in the ecology of the disease is unknown, but it seems unlikely that it plays an important role.

It is somewhat surprising that none of the rodents gave positive protection. Most of these are nocturnal and so would not often be exposed to *Aedes caballus*. *Rhabdomys* is diurnal but may be too small to attract mosquitos.

In 1951 it was reported that the blesbok on affected farms had lost their young. It is noteworthy then that the only two steenbok bloods tested were not protective. Of course this negative finding does not exclude the possibility that this species is susceptible to Rift Valley fever. During the day and at dusk, the steenbok were found taking shelter in the hills on the farm, relatively far removed from the pans, where the mosquitos were found in great numbers. They thus may have escaped infection but further observations are necessary to determine their susceptibility.

Thus no wild animal, except for the one polecat, was found to have had an infection with Rift Valley fever, and none of their ectoparasites was found to be harbouring the virus.

CONCLUSIONS

From this investigation it may be concluded that in 1953 Rift Valley fever still persisted in the Union of South Africa. How and where the infection is maintained has not been determined and presents an interesting problem for future study.

The important vectors are *Aedes caballus* and *Culex theileri*. The former mosquito has been shown in the present investigation to be capable of transmitting the infection while feeding. This has yet to be demonstrated in *Culex theileri*. These are both common species, indeed the predominant species in the pan veld of South Africa, which is a vast area embracing much of the North-Western Cape Province, the Western Orange Free State and the South-Western Transvaal.

Further studies are needed to define the areas in Southern Africa in which Rift Valley fever has occurred and in which it may now be endemic. Further studies

TABLE IV. RIFT-VALLEY-FEVER MOUSE-PROTECTION TESTS

Human Bloods (Initials)		*Result of Test Survivors/total*	*Interpretation*
E.W.		4/5	positive
J.W.		3/5	,,
N.M.		5/5	,,
B.M.		4/5	,,
Negative control	1	1/5	negative
	2	0/5	,,
Positive control	1	5/5	positive
	2	4/5	,,

TABLE V. RIFT-VALLEY-FEVER MOUSE-PROTECTION TESTS

Animal Bloods Species	*No. Tested*	*Result of Tests*
Rattus (aethomys) namaquensis (golden rat)	2	negative
Otomys unisulcatus (Karroo bush rat)	20	,,
Rhabdomys pumilio (striped mouse)	42	,,
Rattus (Mastomys) natalensis (multimammate mouse)	6	,,
Desmodillus auricularis (Namaqua gerbil)	1	,,
Mus musculus (house mouse)	1	,,
Elephantulus myurus (elephant shrew)	5	,,
Xerus inauris (ground squirrel)	5	,,
Cynictis penicillata (yellow mongoose)	6	,,
Ictonyx striatus (muishond)	1	positive
Suricata suricatta (suricate)	1	negative
Felis nigripes (blackfooted cat)	1	,,
Lepus capensis (Cape hare)	1	,,
Pedetes capensis (spring hare)	16	,,
Raphicerus campestris (steenbok)	2	,,

TABLE VI. RIFT-VALLEY-FEVER MOUSE-PROTECTION TESTS

Bird Bloods Species	*No.*	*Result*
Bubulcus ibis (cattle egret)	1	negative
Paccillonitta erythrorhynca (red-billed teal)	1	,,
Ardea melanocephala (heron)	1	,,
Fulica cristata (coot)	9	,,
Alopochen aegyptiarus (Egyptian goose)	1	,,

are also needed to determine what part, if any, is played by wild animals and their ectoparasites in the ecology of this infection in South Africa.

SUMMARY

Rift Valley fever, which was first recognized in South Africa in 1951, again caused an epizootic among sheep in the South-Western Orange Free State in the autumn of 1953. As in the first outbreak several cases of human infection occurred amongst the farmers and the farm labourers who handled the meat or viscera of sheep which had died or were killed when sick of this infection.

Aedes caballus and *Culex theileri*, two of the mosquitos most prevalent in the pan-veld area of this region, were found to harbour the virus. Naturally infected *Aedes caballus* mosquitos were shown to be capable of transmitting the infection whilst feeding.

A large number of the animals and birds were collected

from the affected farms. Sections of the liver from these were examined histologically and the bloods were submitted to mouse-protection tests for Rift Valley fever and their ectoparasites were tested for the presence of Rift-Valley-fever virus by the inoculation of a suspension prepared from them into mice. No lesions of Rift Valley fever were detected in the livers of these animals and birds. All the mouse-protection tests gave negative results for Rift Valley fever except the blood of one *muishond* (polecat) *Iconyx*, which gave a positive result.

Rift-Valley-fever virus was not isolated from any of several batches of arthropods from these animals and birds collected on the farms in the affected area.

REFERENCES

1. Mundel, B. and Gear, J. (1951): S. Afr. Med. J., **25**, 797.
2. Joubert, J. D. S., Ferguson, A. L. and Gear, J. (1951): *Ibid.*, **25**, 890.
3. Gear, J., De Meillon, B., Measroch, V. and Davis, D. H. S. (1951): *Ibid.*, **25**, 908.
4. Schrire, L. (1951): *Ibid.*, **25**, 926.
5. Freed, I. (1951): *Ibid.*, **25**, 930.

RELAPSING FEVER IN SOUTH AFRICA WITH A RECORD OF ITS OCCURRENCE IN EUROPEANS

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Relapsing fever is characterized by pyrexia of sudden onset lasting 3-5 days with rapid subsidence of temperature, followed by a number of relapses at intervals of 1-7 days or longer. The disease is caused by spirochaetes which are present in the circulating blood of the patient during the pyrexial period and in the internal organs when the temperature has dropped to normal. With each relapse the spirochaetes return to the blood stream and may readily be detected by blood-smear examination made at the rise of temperature. Diagnosis of the infection in the apyrexial period is possible by biological tests involving the intraperitoneal inoculation of mice or by a complement-fixation test with the suspension of spirochaetes as antigen.

Two varieties of relapsing fever are distinguished according to the vector of the spirochaetes. Louse-borne relapsing fever is caused by the spirochaete *Borrelia obermeieri* (*recurrentis*) and is met with extensively in Eastern Europe, North and West Africa, India, and the southern parts of the United States of America. Tick-borne relapsing fever occurs in Persia, Central and South America, Spain, Central and South Africa and elsewhere; it is spread by various species of the Ornithodorus group of ticks and in Southern and Central Africa is caused by *Borrelia duttoni*.

In South Africa the tick *Ornithodorus moubata* is the vector. It is found in the cracks and crevices of the walls and floors of the inferior types of dwelling occupied mainly by Natives. The ticks remain hidden during the day and emerge at night to suck blood from their sleeping victims. The spirochaetes which are present in the secretions and excretions of the infected ticks enter the tissues through the bite.

The distribution of the tick and of the disease in South Africa was described more than a decade ago.[1] Present information with regard to the disease is shown graphically in the accompanying map (Fig. 1).

The shaded portions of the map indicate the districts in which cases have been reported. It will be seen that relapsing fever is widely distributed in the Northern,[2] Eastern and Western Transvaal.[1] It also occurs in the Northern part of the Cape Province,[3] as well as in the Graaff-Reinet [4] and Kimberley [5] districts.

The disease and the tick vector are probably more widely distributed than here indicated and it is likely that cases of the infection have escaped recognition.

It will be observed from the map that relapsing fever in South Africa is found mainly on its northern border adjoining Bechuanaland and Southern Rhodesia and on its eastern border adjoining Portuguese East Africa. Relapsing fever is endemic in these countries, whence the disease has spread to the Union. It is interesting to note that the disease appears to have travelled down the central semi-arid regions of South Africa *via* Kimberley through to the Jansenville district within a hundred miles of the coast on the south. The reason for this is not clear but it must be assumed that the disease was spread through the agency of Natives trekking south through these hot dry regions eminently suitable for the development of the tick.

In Johannesburg and the Reef, relapsing fever has since 1934 not infrequently been reported in 'tropical' Natives imported from Nyasaland and other territories from Central Africa as labourers on the gold mines of the Witwatersrand. These Natives, who had undoubtedly acquired their infection in their tropical home-towns, periodically suffer from relapses of a mild type whilst in this country.

RELAPSING FEVER IN EUROPEANS

The Europeans in the endemic relapsing-fever areas in South Africa generally live in houses of good construction and it is thus not surprising that European cases of this disease are uncommon. A European could conceivably develop relapsing fever from living in a poorly constructed house harbouring infected ticks, or taking shelter in empty Native huts while travelling, or camping on sites previously occupied by Natives, especially on sandy soil or near bush where ticks are present.

A survey has been made of the records [6] of blood-

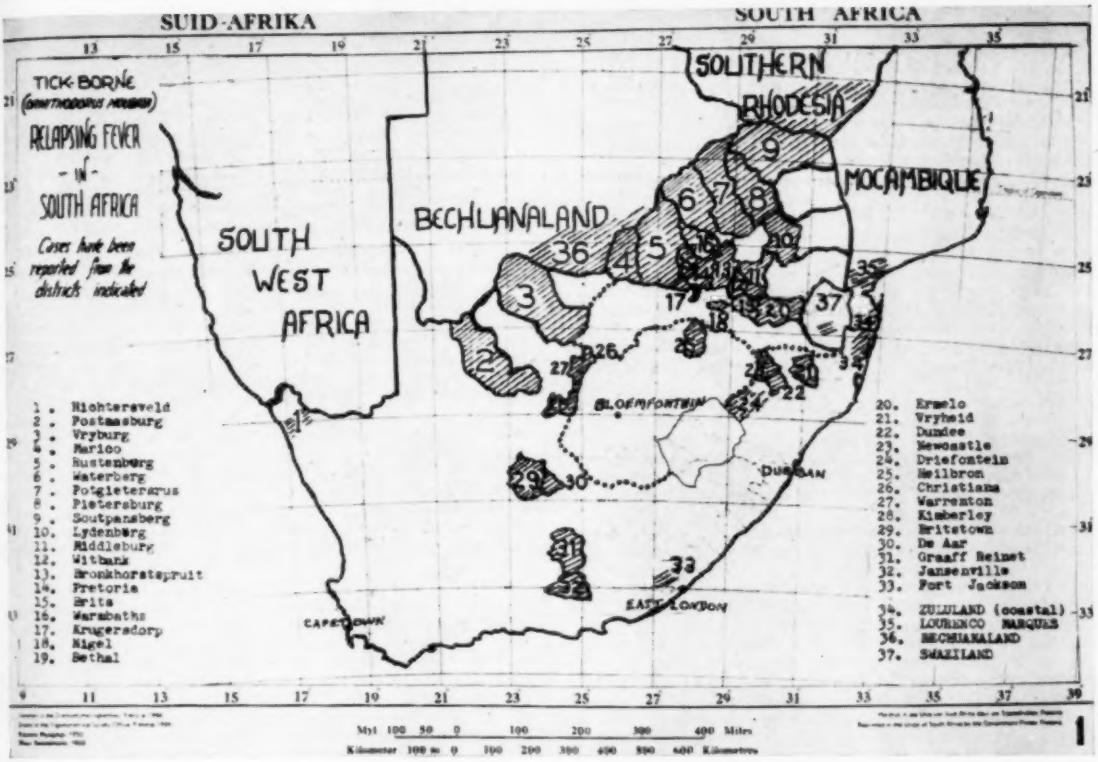

Fig. 1. Tick-borne (*Ornithodorus moubata*). Relapsing fever in South Africa. Cases have been reported from the districts indicated.

TABLE I. RELAPSING FEVER IN EUROPEANS. BLOOD SMEARS POSITIVE FOR SPIROCHAETES REPORTED BY THE SOUTH AFRICAN INSTITUTE FOR MEDICAL RESEARCH, 1932-1954

Year	*Cases*	*Case No.*	*District*	*Details available*
1932	1	1	Potgietersrust	Nil
1933	0			
1934	0			
1935	0			
1936	0			
1937	1	2	Rustenburg	Nil
1938	0			
1939	2	3	Zeerust	Nil
		4	Vryheid	See text
1940	0			
1941	0			
1942	2	5	Johannesburg	Nil
		6	Trichardt	See text
1943	1	7	Johannesburg	Nil
1944	0			
1945	2	8	Nigel	Nil
		9	Pietersburg	See text
1946	5	10	Britstown	Nil
		11	Groot-Marico	See text
		12	Graaff-Reinet	See text
		13	Lourenco Marques	See text
		14	Rustenburg	See text
1947	0			
1948	1	15	Rustenburg	See text
1949	1	16	Rustenburg	See text
1950	0			
1951	1	17	Bronkhorstspruit	See text
1952	0			
1953	1	18	Warrenton	See text
1954	0			

smear examinations carried out at the South African Institute for Medical Research, Johannesburg, for the past 23 years to determine the incidence of relapsing fever in Europeans. Anything up to 100 blood smears are annually reported as positive but nearly all are from Natives.

In Table I the European cases of relapsing fever reflected in positive blood smears are listed and the districts of probable origin of infection indicated.

Of the 18 positive blood-smears reported in Europeans details are unfortunately lacking in 7 of the cases. The European patients became infected in Potgietersrust, Rustenburg, Zeerust, Vryheid, Trichardt, Pietersburg, Britstown, Groot-Marico, Graaff-Reinet, Bronkhorstspruit and Warrenton. One European acquired his infection in Lourenço Marques while the source of infection in two sufferers who were treated for the disease in Johannesburg could not be determined.

CASE HISTORIES

Case 4. A European male child living in Alberton. The patient visited Vryheid in December 1938, where he was bitten by a 'tampan tick'. He returned to Johannesburg on 1 or 2 January 1939 and became ill a week later with a temperature of 103°F, rapid pulse, severe headaches, vomiting and dyspnoea. He complained of a 'stomach-ache' and a feeling of giddiness. The lungs were clear but there was tenderness over the spleen, which was palpable on inspiration. Albuminuria was present. There was bile in the vomit but jaundice was not noted. A slight rash was present on the abdomen. Epistaxis occurred as the temperature

was subsiding but not during the acute stage. Relapses occurred on 15 and 23 January. Spirochaetes of relapsing fever were seen in a blood smear taken on 24 January.

Case 6. A European female, Trichardt. Dr. L. Becker reported that the patient was infected with relapsing fever in the Bushveld in the Loskop Dam area, which she visited from 20 to 22 December 1941. She became ill on 5 January 1942 after returning to her home in Trichardt. A blood smear then revealed the presence of spirochaetes of relapsing fever.

Case 9. A European boy 6 years of age living in Johannesburg. Dr. J. Beeder reported that the patient visited Pietersburg from 7 to 20 January 1945. The illness commenced about 23 January. He was seen a week later and during this period had had intermittent attacks of fever with rigors. The spleen was enlarged. As malaria was suspected quinine was administered. A blood smear however showed the spirochaetes of relapsing fever. The fever subsided after a day or two and the patient appears to have had only one attack subsequently.

Case 11. A European male 30 years of age living in Groot-Marico, Transvaal. Dr. C. A. Marais reported that the patient lived in the Groot-Marico district 12 miles from town in the direction of Zeerust in a house of brick walls and wooden floors and ceilings. The patient declared that he was bitten by ticks in the veld. He became ill on 3 January 1946 with a temperature of 103° F. Examination revealed a moderately enlarged liver with some enlargement of the spleen. There was general weakness and slight jaundice. Urine examination was negative. As malaria was suspected quinine therapy was commenced. The patient was well after 4 days but soon fell ill again. The liver was palpable but not the spleen. A relapse occurred on 20 January characterized by a high temperature with rigors and now both liver and spleen were enlarged. A blood smear revealed the spirochaetes of relapsing fever. The patient, who had received NAB injections, was much improved after 2 days but still felt weak.

Dr. Marais had observed that tick bites were fairly common in the Groot-Marico district, where newcomers rather than local inhabitants were affected by them.

Case 12. A European man 34 years of age living in Johannesburg. He visited Graaff-Reinet on holiday from 3 to 29 December 1945, where he lived with his parents. He returned to Johannesburg on 30 December and next morning became ill and remained so for 3 days. He resumed work for a few days but once more had a relapse lasting 3 days.

He was admitted to the Johannesburg Hospital on 31 January 1946 with a temperature which lasted only 1 day, followed however by 6 or 7 relapses. A blood smear positive for the spirochaetes of relapsing fever was reported on 3 February. Each relapse was characterized by pyrexia, rigors and perspiration followed by headache and general weakness after the acute stage. The liver and spleen were enlarged during these attacks but jaundice was absent. A further relapse occurred on 14 February with severe headaches, sweating and drowsiness. Jaundice was now present with enlargement of the spleen and liver. The patient had had continuous headaches since the onset of the illness with aching of the eyes and severe aching of the back of neck, hip and legs.

Case 13. A European boy 16 years of age living at Christiana in the Transvaal but attending school in Potchefstroom. The patient became ill on 21 September 1946. There was no history of a tick bite at any time, including the period of a camping holiday at Lourenço Marques from 3 to 24 July of that year. From 23 September onwards he developed a headache with afternoon temperatures of 101-103° F for about a month. Apart from epistaxis there was no other complaint. The spleen was not enlarged. On 30 September the temperature was still high and the patient was perspiring freely. A blood smear was negative for relapsing fever but biological tests carried out on the patient's blood were positive for relapsing-fever infection. On the following day the temperature became normal and remained so.

Case 14. A European man 63 years of age—a farmer in the Rustenburg district. He became ill on 25 December 1946. He stated that he had been bitten by a 'big tick' between the toes while working in his tobacco lands on 5 December. He was admitted to hospital on 21 February 1947, after having suffered 8 relapses since his first attack. A blood smear on 28 February was positive for relapsing-fever spirochaetes.

Case 15. A European boy 8 years of age living in the Rustenburg district. He had been bitten by ticks at his home on 4 July 1948 and fever commenced 10 days later, lasting about 12 days. A relapse occurred 2 weeks later. He was admitted to hospital on 22 August with pyrexia which lasted 4 days, followed by another 5 days thereafter. Blood-smear examination showed spirochaetes of relapsing fever.

Case 16. A European man 64 years of age—a retired schoolteacher in the Rustenburg District. He became ill on 7 February 1949 with a temperature of 103° F, which returned to normal in a few days. He was admitted to hospital on 16 February with a relapse. A blood smear taken the following day was positive for spirochaetes of relapsing fever. The patient was unaware of having been bitten by a tick but admitted the possibility of it.

Case 17. A European boy 3 years of age living near Bronkhorstspruit in the Transvaal. He first became ill on 7 December 1950 with pyrexia, rigors, delirium, headache and pain in the legs, which lasted 3 days. He had 3 relapses subsequently with intervals of apyrexia of 3 or 4 days. When first seen by Dr. C. Zaayman the child was in his 4th relapse, with a temperature of 104° F. There was considerable enlargement of the spleen and blood smears showed the presence of relapsing-fever spirochaetes. By courtesy of Dr. Zaayman I paid a visit to Bronkhorstspruit to see the patient in his home. The house, about 25 years old and in a delapidated condition, had walls of unplastered brick and stone. The mother and 3 children were living in the house under unhygenic conditions. All appeared in good health at the time except the patient, who was in bed with high fever. The mother stated that the other children had at various times been similarly affected with this type of illness. Numerous *Ornithodorus moubata* ticks were readily collected from cracks in the walls of the patient's room.

Case 18. An unmarried European man 21 years of age living in Johannesburg. He was born in the Wolmaransstad District, where he had lived for 3 years. He spent a portion of his childhood years also in a Northern Rhodesia mining town. From 1949 he lived with his parents on a farm near Warrenton in the Cape Province, where the house was comparatively new, with rough plastered brick walls and wooden floors. The Native servants on the farm lived in brick huts in the grounds. Eight months after arrival at the farm he began to suffer from attacks of fever with headache and vomiting which necessitated his staying in bed for periods of 3-4 days. He arrived in Johannesburg for permanent residence in January 1953, and remained well for 5 months, when he was again attacked by the same type of illness he had experienced at his home. He had suffered 6 relapses in Johannesburg up to September 1953, when he was admitted to Edenvale Hospital under the care of Drs. G. Lange and I. Segal. He complained of headache, abdominal pain and nausea. On admission the temperature was 101° F and tenderness was present in the right hypochondrium. A tentative diagnosis of infective hepatitis was made, but was not confirmed by laboratory tests. Investigation of the blood showed a raised sedimentation rate and an absolute monocytosis. The temperature became normal the day after admission and he was discharged from hospital on 6 October, to be readmitted on 2 November with headaches, backache and profuse perspiration. His temperature was slightly over 99° F, but returned to normal on the following day. A relapse occurred on 13 November, commencing with a rigor and a temperature of 103° F. Blood smears were positive for the spirochaetes of relapsing fever. The temperature became normal the following day.

There is little doubt that the patient had originally contracted relapsing fever at his home in the Warrenton district.

The following two cases although not in Europeans (a Eurafrican and an Indian) are placed on record because they do not fall into the Native group:

Case 19. A Eurafrican (Coloured) woman 43 years old. She was infected with relapsing fever while working as a nurse in the Native location in Graaff-Reinet which has already been described [4] as being heavily infested with *Ornithodorus moubata* ticks. Dr. A. L. te Water reported that the patient was bitten by ticks on 16 December 1944 whilst in attendance on a maternity case in the location. The patient had 4 relapses at 7-10 day intervals.

Case 20. An Indian man 20 years old living in Johannesburg. He visited Lourenço Marques from 27 December 1947 to 24 February 1948. There was no history of a tick bite. Symptoms

commenced during his stay in Lourenço Marques in the last week of January 1948. Three weeks before his return from Lourenço Marques he developed fever. After his return to Johannesburg he was admitted to the Coronation Hospital on 11 March with pyrexia which lasted 2 days. The spleen was palpable and soft, but not tender. A blood smear showed the presence of spirochaetes of relapsing fever. The patient had most likely become infected in Lourenço Marques.

In addition to these cases, 5 blood smears from Europeans positive for relapsing-fever spirochaetes have in the last 20 years come to the notice of Dr. S. Annecke [7] of the Union Health Department, Tzaneen. Details of these cases are not available except that they represent 3 men and 2 women all from the Pietersburg district.

The total of 23 cases of relapsing fever over a period of 23 years reflects a very small infection rate in Europeans. The possibility that other cases have in fact occurred but remained undiagnosed cannot of course be excluded.

Physicians should consider relapsing fever in differential diagnosis in the areas shown in the map as well as in adjoining regions in any patient with a recurrent type of temperature, and should attempt to confirm its presence by the examination of blood smears taken at the height of the temperature or enlist laboratory aid for a complement-fixation test on the patient's blood-serum.

SUMMARY

Relapsing fever transmitted by the *Ornithodorus moubata* tick occurs mainly in the northern and eastern parts of the Union of South Africa bordering the countries where the disease is endemic, and also in the semi-arid central regions through Kimberley and Graaff-Reinet.

The infection rate in Europeans in South Africa from relapsing fever (tick-borne) is very low; only 23 cases are on record as having occurred in the last 23 years. Other cases in Europeans may have escaped notice, and physicians in the endemic areas are well advised to bear in mind the possibility of a diagnosis of relapsing fever in cases where pyrexia in a patient is unexplained.

REFERENCES

1. Ordman, D. (1941): S. Afr. Med. J., **15**, 383.
2. *Idem* (1943): *Ibid.*, **17**, 180.
3. *Idem* (1939): *Ibid.*, **13**, 491.
4. *Idem* (1944): *Ibid.*, **18**, 272.
5. *Idem* (1944): *Ibid.*, **18**, 259.
6. Annual Reports S. Afr. Inst. Med. Res., Johannesburg.
7. Annecke, S. Personal communication.

DERMATITIS HERPETIFORMIS TREATED WITH DIAMINO-DIPHENYLSULPHONE (DADPS)

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Dermatitis herpetiformis is a chronic skin disease of unknown aetiology, characterized by intense itching and the presence of grouped erythematous, papulovesicular, vesicular and bullous lesions, which on involution leave pigmented spots and sometimes scars. The disease, which may persist for 10 years or more, has natural remissions and exacerbations.

There is no cure for the disease, but there are several well-tried drugs which control both the rash and the itching. Arsenic was probably the first of these drugs to be used, given either as liquor arsenicalis or as acetarsone. The disadvantage of arsenic is its toxicity, particularly when administered over prolonged periods. Arsenical keratoses with their tendency eventually to develop into squamous epitheliomata are a real hazard to the patient taking arsenic.

Sulphapyridine is also effective in controlling dermatitis herpetiformis, and has probably been the most widely-used drug in this condition. It is, however the most toxic of the sulphonamides, and because of an idiosyncrasy to it, many patients are unable to continue treatment. The treatment of the patient who is unable to take this drug is a difficult problem, and any drugs which are reputed to control the disease are worthy of trial.

SULPHONES

The sulphones, a group of drugs which have been used for some years in the treatment of leprosy and tuberculosis, have also recently been used with good effects in dermatitis herpetiformis.[1, 2] Cornbleet [2] has treated 13 cases of dermatitis herpetiformis with diasone with excellent results; some of these cases were refractory to other treatment, including sulphapyridine. Since it is thought that the more complex sulphones act by liberating diamino-diphenysulphone (DADPS) in the body, it was considered logical to investigate the effect of DADPS on dermatitis herpetiformis, particularly since it has been held by some to be superior to sulphapyridine in controlling the condition.[3] DADPS is better absorbed from the gastro-intestinal track than the more complex sulphones, and blood levels are better maintained with it, since it is the most slowly eliminated of the sulphone compounds.

Toxic effects from its use in leprosy and tuberculosis include dermatitis, lepra reactions, leprous neuritis and iritis. Haemolytic and hypochromic anaemia have also been recorded, as well as jaundice due to hepatitis. The commonest side-effect of sulphone therapy is a

transient normocytic anaemia, which appears during the first few weeks of treatment but clears spontaneously in spite of continued sulphone therapy. Other blood changes include methaemoglobinaemia, which occurred in half of the patients treated by Cornbleet with diasone.

The first cases selected for treatment were those patients unable to tolerate sulphapyridine therapy because of nausea and vomiting or severe leukopenia. Other cases were included in the trial because they did not respond to sulphapyridine therapy and the control of the rash and itching had become a difficult therapeutic problem. The result of this therapy in the first few cases was so impressive that it was decided to treat cases well controlled with sulphapyridine, in order to compare the efficiency of the latter with that of DADPS.

CASE REPORTS

Case 1. H.W., female aged 53 years, suffering from dermatitis herpetiformis since May 1952. She was first seen in August 1952, when she was treated with sulphapyridine, 0·5 g. *t.d.s.* This relieved the irritation, but could not be continued because of nausea, vomiting and weakness. She then underwent various treatments, including liquor arsenicalis, nicotinamide and antihistaminics, with no marked effect. On 5 March 1954 she was started on DADPS, 50 mg. daily, with marked improvement. Two weeks later the dosage was decreased to 50 mg. every second or third day. She has shown no toxic effects and remains completely free of rash and itching. The blood count and haemoglobin level remains normal.

Case 2. E.H., male aged 38 years, suffering from dermatitis herpetiformis since 1947. He was first treated with sulphapyridine in November 1950, with marked subjective and objective improvement, but had to be discontinued after several weeks because the patient developed an acute tonsilitis associated with a leukopenia (white-cell count 3,000 per c.mm., with differential count: polymorphs 54%, lymphocytes 36%, monocytes 8%, band forms 2%). In November 1954 sulphapyridine, 0·5 g. daily, was again given, and in 2 weeks the white cell count fell from 7,000 per c.mm. to 3,000 per c.mm. (normal differential counts). Sulphapyridine was therefore again discontinued. On 15 February 1954 he was put on 50 mg. daily. He was seen again after 2 weeks, when there were no signs of active disease and pruritus had disappeared. Because of a relapse, the dosage was later increased to 100 mg. daily, which completely controls the disease. He shows no toxic effects, and the blood count remains normal.

Case 3. J.P., female aged 2½ years. First seen on 14 February 1954 with a 2 weeks' history of a rash. There are 5 other children in the family, none with any skin disease. The rash was typical of dermatitis herpetiformis, with blisters and bullae showing an annular arrangement. It affected the vulva, thighs, back, face and limbs. She was first treated with sulphapyridine 0·5 g. daily as an out-patient, but there was no response to treatment and she was therefore admitted to hospital. Here, in spite of continued sulphapyridine therapy and liquor arsenicalis, she did not improve. On 27 March 1954 DADPS 25 mg. daily, was given, with improvement noticeable after 3 days of treatment. She developed a microcytic anaemia while on this dosage, but this has improved and continues to improve though the drug is still taken. The rash is not completely controlled on this dosage, and she still develops new bullae, but whenever the drug is discontinued a very severe relapse occurs. She has shown no serious toxic effects, though on occasions she has had as much as 50 mg. of DADPS a day.

Case 4. J.R., male aged 50 years, suffering from dermatitis herpetiformis for the last 2 years. He had been treated for the past 18 months with sulphapyridine, 3 g. daily. The rash had fluctuated during this period without complete freedom from itching and active signs of disease. He had a past history of a perforated duodenal ulcer in 1943, gastrectomy in 1948, and haematemesis after aspirin in 1951. On 10 February 1954 he still showed active lesions of dermatitis herpetiformis on the elbows, scratched papules, scars and erythema on the sacrum. General examination showed no abnormality. Blood-count normal. He was admitted to hospital and given inert tablets (calcium lactate) for 3 days with no effect on the rash. He was then treated with DADPS, 100 mg. daily, and showed remarkable subjective and objective improvement. He continues to take DADPS daily, with excellent control of the rash.

Case 5. H.C., male aged 36 years, suffering for the last 3 years from dermatitis herpetiformis, which had been fairly well controlled by sulphapyridine, 1 g. 3 times a day, though the patient still had active disease as shown by itching and the presence of vesicles. On 6 April 1954 the sulphapyridine was discontinued and DADPS 50 mg. daily substituted. On this dosage he developed a severe relapse and the dosage was increased 1 week later to 100 mg. daily, but this was insufficient to control the itching and the eruption. On 20 April treatment was then changed back to 3 g. of sulphapyridine a day, which again controlled the rash. On 27 April the sulphapyridine was again stopped and DADPS given, 200 mg. a day. The rash is very well controlled on this dosage, though he still shows vesicles. He states that he much prefers DADPS because of the greater relief of itching. Haemoglobin 80%, and examination of the blood shows methaemoglobinaemia.

Case 6. J.B., male aged 63 years, suffering for 7 years from typical dermatitis herpetiformis affecting the shoulders, elbows and scalp, and with marked pigmentation of the skin over the sacrum. On 29 April 1954 DADPS, 100 mg. daily, was commenced with complete relief from itching and no signs of active disease after 2 weeks of treatment. He continues to take 100 mg. DADPS a day for complete control of the eruption. There is no anaemia and no toxic effects from DADPS have occurred.

Case 7. M.A., female aged 31 years, with dermatitis herpetiformis of 4 years' duration. Since February 1951 she had been treated with sulphapyridine, 2 g. daily, but without complete control of the rash or itching. On 29 March 1954 DADPS, 50 mg. daily was commenced and by 5 April 1954 she was markedly improved: the blood count remained normal. On 13 May 1954, DADPS was increased to 100 mg. a day because of the presence of active lesions. In spite of the presence of new lesions, she states that the itching has completely disappeared and 'I can sleep now'. She has shown no toxic effects.

Case 8. L.W., male aged 36 years, with very severe dermatitis herpetiformis for the last 7 years. Attempts had been made to control the rash with sulphapyridine, but these had been discontinued because of severe relapses. He had also undergone prolonged treatment with liquor arsenicalis, which was discontinued when the patient developed arsenical keratoses of the palms. Thereafter he had had antihistaminics, nicotinamide and suramin. In January 1954 DADPS 50 mg. daily was commenced, which soon relieved the itching. The dose was increased after 3 weeks to 100 mg. a day. The patient stated that this treatment had been more effective in relieving the itching than any of the previous treatments, though he still showed severe active skin lesions. This patient suffered also from bronchiectasis, mitral stenosis with congestive cardiac failure, chronic nephritis and arsenical keratoses. On 13 May 1954 he became jaundiced and showed severe cardiac failure. Though there were signs of active dermatitis herpetiformis, he had very little itching. He died on 18 May 1954.

Case 9. A.B., male aged 34 years, with 3 years' history of dermatitis herpetiformis. He had undergone various treatments including liquor arsenicalis, suramin, nicotinic acid and sulphapyridine. The rash was comparatively well controlled by sulphapyridine, 2 g. daily. On 10 March 1954 treatment was changed to DADPS, 100 mg. daily, later increased to 150 mg. a day. The patient states that he feels much better on these tablets and sleeps better because of the absence of itching. He continues to take 150 mg. daily as a maintenance dose and has shown no toxic effects.

Case 10. S.E., male aged 42 years, with dermatitis herpetiformis of 2 years' duration controlled only moderately well by 2·5 g. of sulphapyridine daily. On 9 April 1954 DADPS, 50 mg. daily, was substituted and he relapsed within a few days, so that the sulphapyridine was again given in the same dosage. On 7 May 1954 he still had active vesicles, and 200 mg. of DADPS daily was commenced. This produced complete control of the rash and itching. He shows no active disease after 3 months on 200 mg. daily as a maintenance dose. He much prefers these tablets to sulphapyridine, as the latter made him feel 'off colour'. His blood-count remains normal. He has shown no toxic effects.

Case 11. A.B., male aged 37 years, seen on 9 April 1954 with a 4 months' history of typical dermatitis herpetiformis affecting the beard, axillae, elbows and sacral area. He was given sulphapyridine 2 g. daily with marked relief, both objective and subjective. On 21 May 1954, in spite of the sulphapyridine he had relapsed, and DADPS, 100 mg. daily, was substituted. His rash has virtually cleared, with complete disappearance of itching.

Case 12. E.S., male aged 67 years, with 8 years' duration of rash very well controlled by sulphapyridine, 0·5 g. every alternate day, though occasional itching and vesicles were present. On 7 May 1954 treatment was changed to DADPS 100 mg. daily. By 21 May the rash was still well controlled. The patient states that as far as he is concerned there is no difference between these tablets and the previous ones. He continues to take DADPS 50 mg. daily as a maintenance dose. His blood-count and haemoglobin remain normal.

Case 13. C.W., male aged 51 years, with dermatitis herpetiformis for the last 15 years. He had undergone occasional arsenical therapy and since 1951 had taken 0·5 g. of sulphapyridine daily. On 30 April 1954 he still showed active signs of disease. The sulphapyridine was stopped and DADPS, 100 mg. daily, given. The relief of itching is more complete than with sulphapyridine. He continues to take this dose to control the rash. He has shown no toxic effects.

Case 14. E.G., female aged 63 years, first seen in August 1952 with typical dermatitis herpetiformis. From that time she had been on sulphapyridine, 1·5 g. daily, with comparative comfort and control of the rash. On 6 May 1954 sulphapyridine was stopped, and 3 days afterwards she developed a severe relapse. DADPS 100 mg. daily was started on 20 May 1954 with good control of the rash and itching. She continues on this dosage. The rash is well controlled and no toxic effects have occurred.

Case 15. D.S., male aged 37 years, with rash since 1949. He had been on liquor arsenicalis, 2 m. *t.d.s.*, for one year. In 1950 treatment was changed to sulphapyridine 3 g. daily with no marked response. In April 1954 he was seen with active vesicles, papules, and itching. DADPS was given, 100 mg. a day, and 2 weeks later there was no itching and no signs of any activity. He was last seen on 15 July 1954, when he was taking 25 mg. daily for complete control of the rash. He has shown no toxic effects.

Case 16. W.S., male aged 45 years, with dermatitis herpetiformis since 1947. Treated for 2 years with liquor arsenicalis, 3 m. *t.d.s.* He developed severe relapses when arsenic was stopped. Since January 1949 he has been treated with sulphapyridine 0·5 g. daily. On 14 June 1954 his haemoglobin was 80%, colour index 1·0, and white-cell count 5,000 c.mm., with a normal differential count. DADPS, 200 mg. daily, was then substituted for sulphapyridine, and 6 weeks later there was no itching and complete control of the rash. The patient prefers this treatment to any previous one. There was no change in the blood-counts after 6 weeks of treatment; no abnormal pigments were found on spectroscopic examination of the blood.

Case 17. F.H., male aged 47 years. Since 1950 this patient suffered from typical dermatitis herpetiformis not fully controlled by sulphapyridine, 1·5 g. daily. He was seen on 31 May 1954 with active lesions on the back and scalp, when treatment was changed to DADPS, 100 mg. daily. On 10 June 1954 his haemoglobin was 73%, colour index 0·87, and white cell count 7,000 c.mm., with a normal differential count. DADPS therapy was continued. On 28 June 1954 haemoglobin was 95%, faecal urobilinogen was normal, and examination of the blood showed methaemoglobin present; fragility of the red cells normal. Both the patient and his relatives were alarmed at his appearance, which was due to methaemoglobinaemia, but they were reassured and he agreed to continue treatment. On 5 July 1954 he still had a peculiar colour due to methaemoglobinaemia, but his relatives were no longer concerned about his appearance. His rash remains well controlled on 100 mg. of DADPS a day.

Case 18. E.I., male aged 57 years, with dermatitis herpetiformis of 5 years' duration, only partially controlled by sulphapyridine and later by liquor arsenicalis. When seen on 12 May 1954 he showed active lesions on the scalp, and DADPS, 100 mg. daily, was prescribed. On 29 July 1954 he had no itching, no signs of any activity, and no toxic effects. He continues to take 100 mg. a day as a maintenance dose.

Case 19. F.C., male aged 69 years, with 3 years' history of rash well controlled with sulphapyridine, 1 g. daily. On 10 June 1954 his blood count showed an eosinophilia, and the haemoglobin was 102%. DADPS, 200 mg. daily, was given. On 21 June 1954 he complained of a constant headache, he was very cyanosed and blood examination showed sulphaemoglobinaemia. All treatment was stopped. By 10 July 1954 his cyanosis had disappeared, but the rash had relapsed. DADPS 100 mg. daily was given, and on 26 July 1954 the patient again complained of severe headaches. He was again cyanosed, and sulphaemoglobin was again present on spectroscopic examination of the blood. The DADPS was continued and ascorbic acid, 900 mg. daily, was given. As the cyanosis persisted on this treatment, the DADPS was discontinued.

RESULTS OF TREATMENT

The 19 cases of dermatitis herpetiformis treated with DADPS include one case of the infantile type of the disease. All these cases responded to treatment. The relief of itching and the disappearance of the rash was quite dramatic in most cases. The dosage of DADPS appears to be directly proportional to the dosage of sulphapyridine necessary to control the eruption. No case required more than 200 mg. of DADPS a day to control the rash, the average maintenance dose being 100 mg. a day. As with sulphapyridine, relapses generally occur on the 3rd day after the patient discontinues treatment. Since the disease is characterized by spontaneous remissions and exacerbations, patients were told to modify their dosage as they found necessary. Only one patient (case 12) noted no difference between DADPS therapy and sulphypyridine. All the other patients preferred DADPS therapy because of the greater relief from itching and the absence of side effects.

TOXIC EFFECTS

Kruizinga and Hamminga [3] noted no severe toxic effects in their 12 cases, nor were there any present in our 19 cases.

One patient (case 19) was obliged to discontinue treatment because of sulphaemaglobinaemia associated with severe headaches. This did not respond to big doses of ascorbic acid, so DADPS was discontinued.

Two cases showed methaemoglobinaemia. These continued with their DADPS therapy and had no other toxic effect from this drug.

One patient (case 8) when first seen, was very ill, suffering from bronchiectisis, mitral stenosis and chronic nephritis. Though he obtained substantial relief of itching from DADPS, he continued to show large numbers of vesicles and blisters. This may have been due to insufficient dosage of the drug, and the continued absorption of iodine from the lipiodol in his bronchiectatic cavities. Though he was jaundiced for 2 weeks before his death, the jaundice could not be attributed definitely to the DADPS.

DISCUSSION

The mode of action of DADPS in dermatitis herpetiformis is unknown. The more complex sulphones, e.g. diasone and sulphatrone [1, 2] have been used in the treatment of dermatitis herpetiformis with favourable results, though their action is generally inferior to that of sulphapyridine. As these more complex sulphones are thought to be broken down in the body to DADPS,

it is understandable that DADPS should also have a favourable action in dermatitis herpetiformis.

SUMMARY

Nineteen cases of dermatitis herpetiformis were treated with DADPS for an average period of 3½ months. All cases responded to treatment provided dosage was sufficient. The relief of itching and the control of the rash is more pronounced with DADPS than with sulphapyridine.

I wish to thank Drs. H. R. Vickers, I. Sneddon, D. Fletcher, R. Church and M. McElligott of the Rupert Hallam Department of Dermatology, Royal Infirmary, Sheffield, for access to their cases and for their advice.

REFERENCES

1. Esteves, J. and Brando, F. N. (1952): Ann. Derm. & Syph. (Paris), **79,** 224.
2. Cornbleet (1951): Arch. Derm. Syph. (Berlin), **64,** 684.
3. Kruizinga, E. E. and Hamminga, H. (1953): Dermatol., **106,** 387.

THORACIC OESOPHAGEAL DIVERTICULUM

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This is neither an article on a known though comparatively uncommon condition nor a report on a series of cases. The case reported however presents etiological and clinical features that warrant reporting it.

Oesophageal diverticula are classified into pharangeal, thoracic and epiphrenic according to locality. Etiologically again they are divided into traction or pulsion depending on the causative factor present. Boyd[1] thus stresses the point that, as in hernias and aneurysms the diverticulum owes its origin to pressure from within or traction from without.

To the writer's mind this theory holds good for pulsion only in the case of cervical (pharangeal) diverticula, since in that region no traction is present.

Thoracic oesophageal diverticula, i.e. those below the bifurcation of the trachea, are usually due to traction exerted by old tubercular lesions, whereas epiphrenic ones, i.e. those just above the diaphragm, are true pulsion diverticula due to obstructed deglutition caused by cardiospasm, or a low oesophageal obstruction caused by the scar tissue resulting from syphilis, tuberculosis, or a high gastric ulcer involving the oesophageal entrance into the stomach. These diverticula are small—often multiple. Lindskay and Liebow[2] consider an already existing weakness of the oesophageal wall as the main causative factor. Sellors[3] states that although pulsion diverticula are fairly common phenomena they practically always occur at the upper end of the oesophagus, being so rare in other parts of the oesophagus that they can safely be disregarded.

Mogendie[4] found 10% of oesophageal diverticula to be thoracic but does not state whether these cases were mid-thoracic, like the case presently to be described, or epiphrenic. Lahey and Warren[5] report that out of a series of 374 cases there occurred 9 epiphrenic pulsion diverticula. Here again a truly thoracic one is not mentioned.

The case to be reported was interesting etiolotically in that it conformed to neither of the etiological theories advanced by the aforementioned authors. There was no pulsion or traction factor present, nor was there a history or clinical evidence of lues or tuberculosis or obstruction lower down. Professor Saint[6] however advanced the very interesting theory that where there has been a large intake of alcohol over a prolonged period of time an inflammatory reaction of the nerve supply of the oesophagus sets in. There is a resulting weakness of the oesophageal musculature followed by diverticulum formation. The case reported conforms to this theory.

CASE HISTORY

K. was kindly referred to me by Dr. P. Brink. The patient was complaining of a continuous feeling of nausea followed by copious vomiting approximately 2 hours after a meal. Two years previously he had only noticed epigastric discomfort, but the condition had deteriorated till he now was forced to seek medical advice.

The past history was good except for an intake of alcohol well above usual extending over the past 6 or 7 years. There were no serious illnesses or operations.

Physical examination revealed a fairly well nourished male aged 46 years. Except for slight deafness all the other findings were within normal limits.

Considering his age and the symptoms complained of, peptic pathology was the first consideration and he was referred to Drs. Le Roux and McCallum for a barium meal. Only one plate of an excellent series of pre-operative films is here reproduced (Fig. 1) and the diverticulum is shown to be mid-thoracic, that is to say the ostium, although owing to its size the belly is practically resting on the diaphragm and partly posterior to the heart, is just below the bifurcation of the trachea.

Operative treatment was decided on. Since the blood supply of the oesophagus is none too copious it was difficult to decide on the best operative approach, since simple extirpation would leave a very wide defect in the oesophagus and thus a great possibility of post-operative sloughing of the suture line with the complications following an intrathoracic leakage.

A very feasible alternative would be anastomosis of the diverticular pouch through the diaphragm with the stomach. This would be fairly easy technically

Fig. 1

but subsequent regurgitation of acid stomach contents into the pouch might then cause anastomatic ulcer formation.

The last alternative would be amputation of the oesophagus above the diverticulum and anastomosis with the stomach drawn up through the diaphragm into the thorax.

Since the last measure would be quite a formidable operation equivalent in magnitude to an abdomino-thoracic total gastrectomy with all its possible complications, extirpation of the sac was decided on as probably being the lesser of many evils.

The thoracic cavity was entered through the 10th interspace after removal of the 9th rib. Adhesions or other signs of previous intra-thoracic pathology were conspicuous by their absence.

Oesophagus and vagi were identified but the diverticulum was nowhere to be seen. However, after the pulmonary ligament had been split, which enabled the assistant to lift the lower lobe of the lung upward and at the same time displace the heart to the left, the diverticulum was exposed in its entirety.

The sac was lifted from its bed and put under tension —slight since it was found to be friable and started bleeding where touched by the forceps. The diverticulum was now amputated but *not* too flush with the oesophagus wall, so as to leave a liberal edge which could be folded inwards and thus avoid future stricture formation. Mucosa was separated from media and serosa and the two cut edges joined with a continuous catgut no. 00. Media and serosa were now joined in a separate layer and reinforced with interrupted silk stay-sutures.

There was no bleeding throughout the operation. But the old adage has it, 'When in doubt drain', and the thorax was accordingly closed in the usual manner after institution of underwater drainage.

Two pints of blood were transfused during and after operation, although this was hardly necessary since patient's condition remained excellent throughout.

Post-operatively a Ryall's tube was left *in situ* and small feeds given through the tube on the 2nd day. The patient, however, was very restless and on the second post-operative night pulled the Ryall's tube out. Realizing what he had done he tried to put the tube back again, and the resultant noise attracted the attention of the Night Sister! Needless to say the worst was feared after this heroic effort. However, he was none the worse for his attempted co-operation and after liberal doses of C_2H_5OH had been added to the milk he settled down nicely and never looked back again.

He made an uneventful convalescence and after 2 weeks was enjoying a full meal.

Pathological examination of the sac was made by Drs. Clegg and Finlayson who reported as follows:

Specimen Received: A diverticulum removed from the oesophagus. It was flabby and pouch-like having a brown outer surface and a pink colour inside where the mucous membrane

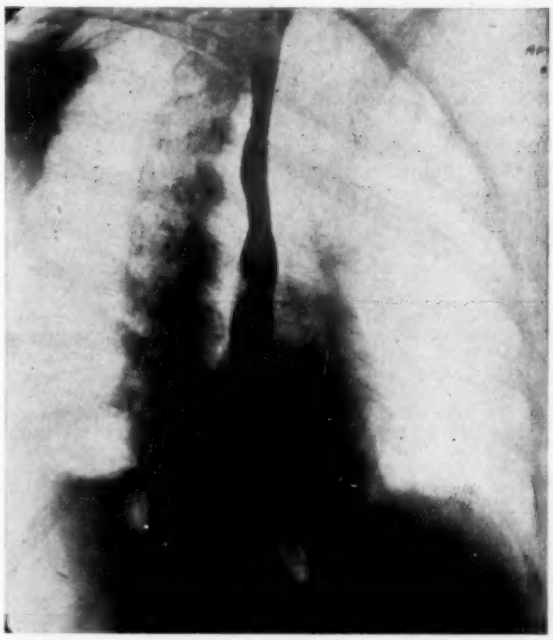

Fig. 2

was situated. There was also a tiny yellow spot where there may have been an ulcer.

Microscopic Examination: The diverticulum is lined by stratified squamous epithelium which appears orderly and benign. It is broken at the place where the yellow spot was seen macroscopically and an ulcer exists but there is very little evidence of active inflammation although the floor is made of very congested connective tissue. The muscularis mucosae is well preserved but the outer muscle walls (both longtitudinal and circular) are practically non-existent, only traces being apparent here and there. The appearances are benign and no evidence of neoplasia could be seen.

Fig. 2 is an X-ray photograph taken 1 month after the operation. At that time the patient was enjoying normal meals and gaining weight.

SUMMARY

The etiology of oesophageal diverticulum is reviewed.

A new theory is put forward, viz. that alcoholic neuritis leading to muscular degeneration is a causative factor.

A case is described which appears to support this theory.

I wish to thank Mr. H. Katz for his valuable assistance at the operation as well as in post-operative care.

REFERENCES

1. Boyd, W. (1947): *Surgical Pathology*. London and Philadelphia: William Saunders.
2. Lindskog, G. E. and Liebow, A. A. (1953): *Thoracic Surgery*. New York: Appleton.
3. Sellors, T. H. (1933): *Surgery of the Thorax*. London: Constable and Co. Ltd.
4. Magendie, J. (1950): J. méd. Bordeaux, **127,** 793.
5. Lahey, F. H. and Warren, K. W. (1954): Surg. Gynec. Obstet., **98,** 1.
6. Saint, C. F.: Personal communication.

SITUS INVERSUS TOTALIS

A REPORT OF A CASE PRESENTING WITH AN 'ACUTE ABDOMEN'

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AND

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Complete heterotaxy, situs inversus totalis or, as this rare condition is more generally known, dextrocardia with complete transposition of the viscera may in the cool quiet atmosphere of the routine clinical examination provide a source of diagnostic difficulty, full discernment coming perhaps after radiographic investigation. When, however, the patient first presents as an acute abdominal emergency in the early hours of the morning the problem becomes magnified by the necessity for early surgical intervention with all the implications of accessibility and the difficulty of manoeuvring on a mirror-image of the normal and conventional anatomical arrangement of the abdominal viscera.

CASE REPORT

Mrs. A.T., aged 34 years and the mother of 2 children, had enjoyed perfect health until the present acute episode, which had persisted for 4 hours before medical aid was sought. Two hours after the evening meal she experienced an acute onset of severe colicky pain in the left hypochondrium lasting an hour. The pain persisted with a waxing and waning in intensity and she was still in obvious distress when she was seen. She felt extremely nauseous and bloated, suffering repeated belching. Recognizable food was vomited on 2 occasions and 2 loose bowel actions occurred during this time. The past history was irrelevant; her appetite had always been good, without disinclination for fried or fatty foods. Her menstrual history was normal.

She was a thin, young and anxious-looking patient, with a temperature of 99 F, pulse rate of 100 per minute and blood pressure of 140/90 mm. mercury. The tongue was clean and moist and there was no evidence of icterus. Routine examination of the chest was carried out and on auscultation of the heart it was startling to note the absence of heart sounds in the left chest, whilst clearly audible sounds emanated from the right praecordium. The absence of tracheal displacement and of signs of pulmonary mischief made a diagnosis of dextrocardia very probable.

Abdominal examination disclosed a tender mass in the left hypochondrium which moved on respiration, and abdominal rigidity was localized to the left upper rectus. Vaginal and rectal examination demonstrated nothing abnormal and it was decided to explore the abdomen for acute left-sided calculous cholecystitis, the possibility of acute appendicitis in an undescended left-sided organ also being considered.

An upper mid-line incision was performed and inspection disclosed the visceral anatomy to be a complete mirror-image of the normal. The stomach, spleen and splenic flexure of the colon were situated in the right hypochondrium, the mesentery ran from right to left and the caecum and appendix were situated in the left iliac fossa. The lesser liver lobe was on the right, the main hepatic organ with the biliary apparatus being situated in the left hypochondrium.

Cholecystectomy was performed for acute calculous cholecystitis, the anatomy of the cystic artery, cystic and common bile ducts being a perfect mirror-image of the accepted normal without any variations. Appendicectomy was also performed and the abdomen was sutured with drainage of the gall-bladder bed. The patient made an uneventful recovery and was discharged from hospital in fit condition on the 8th post-operative day. Examination of the gall-bladder disclosed that cholesterosis had served as the basis for the two large cholesterol stones which were contained within it.

COMMENTARY

Though dextrocardia may exist alone, it is usually associated with complete transposition of all the thoracic and abdominal viscera, the structures retaining a perfect mirror-image relationship to each other. The heart is structurally and functionally healthy, the electro-

cardiogram demonstrating reversal of all the complexes in lead 1 whilst leads 2 and 3 are interchanged.

Kartagener (1933) described the association of complete heterotaxy bronchiectasis and congenital abnormalities of the paranasal sinuses manifested usually by absence of the frontal sinuses. Subsequent investigation of our patient did not disclose the existence of the other features of the triad which is generally known as the Kartagener complex.

Though there have been isolated case reports of the surgical abdomen associated with situs inversus there has been very little discussion regarding the relationship between the clinical features of visceral inflammation and the situation of the transposed viscus—Levering (1945), Broster (1944), Jooste (1945).

The autonomic nervous system provides the visceral sensory mechanism, the ill-localized referred sensation of pain, however, depending on the projection of stimuli along the corresponding somatic nerve by antidromic impulses from the appropriate spinal segment. Once irritation of the overlying peritoneum occurs the pain becomes localized over the affected viscus, as the peritoneum has a somatic sensory nerve supply. Muscular rigidity then develops as a viscero-motor reflex, whilst tenderness and hyperaesthesia may be construed as a viscero-sensory reflex. It is unnecessary for the purpose of this discussion to complicate the issue by considering the phenomena of facilitation or the concept of the internuncial pool —Lewis (1942), Good (1952), Lorente (1938).

If situs inversus totalis is accompanied by transposition of the nervous pathways then the viscero-neural pattern will remain constant, the clinical features of pain, tenderness and rigidity bearing a direct relationship to the site of the affected viscus. As the development of the nervous pathways is not necessarily related to the visceral development, it is conceivable that the visceral transposition may be unassociated with a similar change in the neural connexions. Under such circumstances it must be presumed that the clinical features would be ordained not by the visceral arrangement but by referred mechanisms to the site of its conventional anatomical situation.

REFERENCES

Broster, L. R. (1944): Brit. J. Surg., **31**, 393.
Good, M. G. (1952): Lancet, **72**, 482.
Jooste, E. (1945): S. Afr. Med. J., **19**, 388.
Kartagener, M. (1933): Beitr. Klin. Tuberk., **83**, 489.
Levering, J. W. (1945): Clinics, **4**, 867.
Lewis, T. (1942): *Pain*. New York: The Macmillan Co.
Lorente de No, R. (1938): J. Neurophysiol., **1**, 207.

MINISTER OF HEALTH REPLIES TO MEDICAL MEMBERS IN PARLIAMENT

FROM A PARLIAMENTARY CORRESPONDENT

The Senate's debate on the policy of the Minister of Health, Mr. J. F. Naudé, (reported in the *Journal* 21 May p. 504) was followed within a short time by comprehensive discussions of the Minister's votes in Committee of Supply in the House of Assembly. At the outset of his first intervention in these discussions the Minister said he realized his new portfolio would be a difficult one for him as a layman, but he relied on the support that he knew he could expect from the medical profession.

In discussing the anti-poliomyelitis vaccine, he said it had been gratifying to receive the tribute from America that a great debt was owed to the South African Poliomyelitis Research Foundation for its contribution of scientific information. Before the vaccine would be used in South Africa it would be tested 'doubly and doubly and yet again doubly'. The supply of vaccine would be sufficient for about 250,000 people, and the committee concerned with mass inoculation would advise what age-groups and what areas it would be advisable to tackle first. Every month a further 250,000 persons could be vaccinated, provided sufficient monkeys were obtained.

Dr. C. de Wet, M.P. for Vereeniging District, complimented the Minister on his energetic handling of the poliomyelitis epidemic. He said that, notwithstanding what America had done in producing the anti-poliomyelitis vaccine, South Africa stood with Sweden, Australia and France in the front line of the campaign against this disease, and South African research workers had achieved world fame. It was not improbable that the South African vaccine would be more effective and simpler than even the Salk vaccine.

Millions of parents were uncertain and worried about having their children inoculated, largely because of the inappropriate way the success of the Salk vaccine had been announced in America. 'I do not think the method was generally worthy of the medical profession', he said. 'The manufacturers exploited the worry and concern of humanity to obtain cheap publicity—for a vaccine which still is certainly not fully effective and which will have to undergo intensive tests for many years yet.'

He said there was a tremendous difference between the ethical standards of the medical experts in the South African department and those possibly in other countries. He hoped the high standard in this country would be maintained. He urged the Minister to see that many thorough tests were carried out before mass inoculations began in the Union. It was a strange vaccine and one did not know whether children who were inoculated might be sterile when they grew up. He did not think there was any hurry. Poliomyelitis was a most dramatic illness but it was not reaching anything like the proportions of, for example, diphtheria. In 1953 there were 298 deaths from diphtheria and 247 last year, compared with 193 from poliomyelitis in the 1948-49 epidemic and 35 last year. Emphasising the importance of anti-diphtheria inoculations, he said that if every child in South Africa could be inoculated tomorrow the disease would be eradicated from South Africa within a few years.

The Minister appealed to members of the public to have all children inoculated against diphtheria. It had not been desirable to carry on with these during the recent poliomyelitis epidemic, but now during the winter months the inoculations were perfectly safe. It was urgently necessary to go ahead with them at once.

HOSPITAL ACCOMMODATION FOR MENTAL PATIENTS

In reply to various members who enlarged on the lack of hospital accommodation for mental patients, Mr. Naudé agreed that the present overcrowding was pathetic. Steps were being taken throughout the country to provide better accommodation as far as possible. All the mental hospitals were short of staff. One solution might be to use more Coloured and Native aids instead of having a majority of European nurses. It was unfortunate but unavoidable that a considerable number of mental patients had to be detained in prisons or police cells from time to time. It happened mainly at the smaller centres where there was no institutional accommodation. The man who was certified had to be held somewhere. The policy had obtained for 10 or 20 years, but conditions were being improved as quickly as possible.

Dr. Z. J. de Beer, M.P. for Maitland, said he was pleasantly surprised to hear recently that the overcrowding was relatively moderate: there were 18,730 patients in mental hospitals where accommodation was provided for 17,582. He hoped South Africa would not be slow in following other countries that had trans-

formed their approach to mental disease, regarding it as a disease to be treated like other diseases, with prospects of a cure in many cases. In some of the Union's mental hospitals the admission rate of European male senile dementias was as high as 50 or 60%. This type of case did not require medical treatment, but merely to be kept, fed and washed and to be protected from self-harm. This was in sharp distinction to the more acute forms of mental disease which were treated by electro-convulsive therapy, by psychiatric measures and by a positive approach which effected cures in roughly 40 or 50% of the cases. But all too often this type of case could not obtain admission to hospital until degeneration had set in and they had become incurable.

Many patients of the degenerative type of case of senile dementia and allied disorders blocked for perhaps 20 years beds in mental institutions which might otherwise be used for curable cases. 'It seems to me', said Dr. de Beer, 'that a great deal can be done before we get extra beds, by readjusting the classification of patients and providing the expert treatment facilities which we are fortunate enough to have in certain of our institutions, for those cases where they can be most beneficial'.

The detention of persons in gaol in terms of the Mental Disorders Act meant, apart from other unpleasant considerations, that they could not possibly be given any of the care they deserved, and their condition was bound to deteriorate. He urged that, when mental institutions are sited or moved in future, the extreme importance should be borne in mind of keeping them near enough to medical schools to permit the students to get adequate training in this highly important branch of medicine.

Dr. de Beer quoted from the article which Dr. F. R. Luke wrote in the *South African Medical Journal* last year, about the trend of the provision of medical services, by private physicians to the public, to change in type and in method. As medical science broadened, methods of treatment, unfortunately, tended to become more expensive. So the people of South Africa, as of many other countries, had recourse to medical aid societies. 'It is a development which is regarded by all interested parties, I think, as a healthy one', said Dr. de Beer. 'I would, however, urge the Minister to keep a close eye upon its repercussions on the public and the medical profession, because difficulties are tending to arise which will, unless they can be controlled and dealt with at this stage, wreck the entire scheme in South Africa.'

He quoted from an address on this subject which Sir Earle Page, the Australian Minister of Health, gave to the World Medical Association, when he said: 'The Australian Government believes that a partnership of the medical profession, the community, insurance organizations and the Government can evolve a method of retaining all the existing traditions and advances on the medical side and still bring the cost of a first-rate medical service within the means of the people'.

TUBERCULOSIS, BLINDNESS, DIPHTHERIA, CORONARY DISEASE

Col. O. L. Shearer, M.P. for Pietermaritzburg City—another medical practitioner—said it was psychologically important that a tuberculosis patient on his discharge, when he was no longer suffering actively, should be employed. One of the difficulties in detecting the disease among non-Europeans was the fact that the man ceased to be the breadwinner and the family could not balance its budget.

In speaking about blindness he said that 90% of its incidence in the Union was preventable. It would therefore be a great saving to finance a voluntary agency such as the St. John's Ophthalmic Hospital for the purpose of limiting the incidence. He quoted the words of a doctor who wrote in the *South African Medical Journal* that trachoma 'is of far greater public health significance as a problem requiring urgent control than is poliomyelitis'.

Turning to diphtheria, Colonel Shearer said the country had made tremendous advances in limiting its incidence. Yet the incidence was 30 times as high as in England. It had remained unaltered in the Union for the past 14 years, during which its prevalence in other countries had been reduced by 94 to 100%. To eradicate the disease would require the immunization of between 55 and 80% of the infants and 95% of schoolchildren.

In speaking about cardiac conditions, and particularly coronary disease, Colonel Shearer reminded the committee that Dr. Ancel Keys, a physiologist from the United States, had, during a recent visit to Cape Town, attributed the heavy incidence of coronary disease to a high fat-intake. 'I am not going into the pros and cons of that', Col. Shearer said, 'because I feel that his visit to the Cape was very brief. I do not think that he was justified in making an over-all assertion, particularly in so far as the non-Europeans are concerned. We know that the non-Europeans, especially the Bantu people, have a deficiency rather than an over-intake of fat. But there is no doubt that coronary disease is largely due to a rich diet.'

Mr. Naudé said he happened to attend a lecture by Dr. Keys recently, in which it was shown that most people who suffered from coronary diseases—which were rapidly increasing—had their condition attributed to a large extent to the fact that they ate too much fat. On the other hand there were people who did not have enough fat. More attention would steadily be given to teaching the population to maintain a balanced diet.

SEPARATE REGISTERS

At a later stage of the discussion Dr. de Wet said that of the 7,878 registered medical practitioners and specialists in South Africa, 2,647 were in the service of the Government or the provincial administrations. In other words 1 out of every 3 available doctors rendered free services to the public. One out of every 6 was on full-time service. There was a district surgeon to every 20,000 members of the population, White and non-White. Infectious diseases, for which the Department of Health was responsible, had been brought thoroughly under control.

He did not think that last year's legislation, giving the South African Medical and Dental Council the right to maintain separate registers for various groups of practitioners, created a sound position. But the initiative for a change should come from the Medical Council and the Medical Association of South Africa, as a domestic matter. The onus did not lie with the Minister.

The public was unnecessarily exposed to the payment of higher fees because the specialist, purely through being on a different register of the Medical Council, enjoyed certain privileges over his colleagues of being able to ask for higher fees—without his conduct being properly prescribed and controlled. The only solution appeared to be to allow consultants alone to charge the higher fees.

The Minister said the system in the Union, as elsewhere, was that the wealthy were called upon to pay for the poor. 'I do not think that is so wrong, either', he added. The Government for its part was trying to give the poor the services to which they were entitled.

SUGGESTED SELECTION OF STUDENTS

Mr. D. J. G. van den Heever, M.P. for Pretoria Central, said some universities accepted medical students who were not bilingual, though such persons could not make a success of medical practice in South Africa. They might perhaps do laboratory work instead. He therefore advocated the appointment of a selection board to choose for university entrance only those medical students with the right temperament and the qualifications to work with the broad masses of the people. He added that he had been informed that doctors who served their year's internship in the smaller town hospitals derived good training, but those who went to the large specialist hospitals virtually wasted a great deal of the year. He suggested that the year's work should once more be made subject to inspection.

The Minister replied that the universities had their own rules for the selection of students. Any question of admission would have to be referred to the Department of Education, Arts and Science. He hoped the large hospitals would make provision for newly-qualified doctors to get the necessary further training, even though that was where one found all the specialists. The possibility of appointing an inspector to look into such facilities at the hospitals was being considered, but there was the question of who should pay his salary.

Mr. Naudé urged that more people, Members of Parliament included, should join the blood transfusion services.

ENRICHED BREAD

In answering questions about his Nutrition Vote, the Minister said brown bread was enriched by the addition of a mixture containing ground-nut meal, milk powder, calcium carbonate and calcium acetate. The pre-mix was prepared by a Johannesburg firm and supplied to bakers throughout the Union. The Department of

Nutrition paid £69 7s. 6d. per ton for the pre-mix, and it paid the transport and delivery charges as well. Bakers' fat was added, costing the department an average of £100 12s. 9d. per ton. This fat, supplied to the bakers, was used in the baking process on a basis of 1 lb. per 100 lb. of meal. To every 200-lb. bag of meal was added 13 lb. 6 oz. of pre-mix. The total cost of the enrichment ingredients was 11s. 3d. per bag and the estimated expenditure on the enrichment of bread was now £524,000 a year.

Experiments were continually being carried out on the enrichment of mealie meal, but the problem of discolouring the pure white meal was difficult to overcome. At present the enriched meal was supplied only to prisons and other institutions where people were fed with an improved mixture. The enriching mixture consisted of soya-bean meal, milk powder, food yeast and calcium carbonate. Each 170 lb. bag of mealie meal contained 10 lb. of the enriching mixture, which cost £95 7s. 5d. per ton.

The mines and large industrial concerns were using both enriched bread and enriched mealie meal.

BILLS TO BE DEALT WITH THIS SESSION

FROM A PARLIAMENTARY CORRESPONDENT

The Supplementary Health Services Bill and the Nursing Amendment Bill are among those that will not be proceeded with this year, according to a statement made in the House of Assembly by Mr. C. R. Swart, the Leader of the House. Both Bills have been before select committees since the beginning of this session.

The Friendly Societies Bill remains on the list of those intended to be disposed of this session. It has been reintroduced in the House as amended in select committee. Its interest to the medical profession lies in the fact that it provides for the registration, control and regulation of societies which include in their objects 'the relief or maintenance during . . . sickness or infirmity, whether bodily or mental' of members or their relatives or dependants, and/or 'the provision of or the payment of contributions towards the cost of medical, nursing, surgical, optical or dental attendance or medicines or other medical requirements or surgical, optical or dental appliances or accommodation in hospitals, nursing homes, infirmaries or homes for aged persons' for members or their relatives or dependants.

The House has published the report of the select committee, which inquired into this Bill along with the Pension Funds Bill. It took no oral evidence, but received numerous memoranda and letters, including a number from medical aid societies.

Another Bill intended to be handled this session is the Births, Marriages and Deaths Registration Amendment Bill which the Minister of the Interior recently introduced in the House of Assembly. It has one clause of especial interest to medical practitioners: clause 11, empowering magistrates to issue burial orders when death occurs from unnatural causes.

The burial order shall be issued only if the death is registered, and a death from unnatural causes is registered only subsequent to the post-mortem examination. The clause inserts an addition to Section 24 of the principal Act (No. 17 of 1923), reading: 'In any case not provided for in any of the preceding sections the magistrate shall give an order authorizing burial as soon as he is satisfied that the body in question is no longer required for the purposes of an inquest or other proceeding.'

PASSING EVENTS : IN DIE VERBYGAAN

Dr. Stewart W. Mannion left Cape Town by air on 16 May for Ireland, accompanied by his wife and infant son, Stephen. He and his brother, Dr. Patrick L. Mannion of St. Vincent's Hospital, Dublin, will work together overseas to further their medical studies, before returning to the Cape.

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Scholarships for Medical Students. The Westdene Products Scholarship of 1955, tenable at the Natal University, has been awarded to Mr. B. T. Naidoo, a 4th-year medical student.

* * *

Research Forum Faculty of Medicine, University of Cape Town. A paper on *Hyperventilation Syndrome* is due to be read by Dr. F. Ames at Research Forum on 1 June 1955 at 12 noon in the large A floor lecture theatre, Groote Schuur Hospital.

* * *

South African National Tuberculosis Association. After the preview of the Santa film, 'Meet Mr. Brown,' which is being held at Escom House, Johannesburg, on 8 June at 4·30 p.m., the chairman of the Southern Transvaal Regional Committee of the Buy South African campaign, Major-General Sir Francis de Guingand will address a combined audience of members of the Transvaal Chamber of Industries and the Johannesburg Chamber of Commerce on the close co-operation required from commerce and industry for the pursuance of the anti-tuberculosis campaign.

* * *

The 8th International Pediatric Congress will be held in Copenhagen on 22-27 July 1956 under the patronage of Her Majesty Queen Ingrid of Denmark.

A scientific and technical exhibition will be arranged in connection with the Congress.

The following are members of the Committee of Honour:

Johs. Frandsen, M.B., Medical Director, Professor H. M. Hansen, Ph.D., Principal of the University of Copenhagen, Th. Madsen, M.D., Professor Peter Skautrup, Ph.D., Principal of the University of Aarhus, Agnete Vohtz, Permanent Under-Secretary, Department of Education, Jens H. Zeuthen, Permanent Under-Secretary, Department of the Interior.

The aim of the Congress is to make an international exchange possible of the results of researches into the preventive and curative work during childhood.

The address of the Organizing Committee is Kristianiagade Domus Medica, Copenhagen.

* * *

'Wrapped' Vitamins. Vitamin A deteriorates rapidly in storage but a new technique now 'wraps' each molecule separately, thus preserving the vitamin indefinitely. The vitamin is crystallized in combination with another edible material to form an 'inclusion compound' in which the crystalline structure of the other material encloses each molecule of the vitamin—*American Chemical Society.*

* * *

Growth Inhibitors. Preliminary investigations of chemicals that inhibit the normal growth of insects indicate that there is a great number of these growth-inhibiting chemicals. Small quantities of piperonyl butoxide, for example, added to the bran-yeast mixture in which flies are reared in the laboratory, were found to prolong the larvae-to-adult development period by 2 days. When the amount of chemical was increased to 0·25% by weight of the culture medium, no adults developed. The chemical was more effective against the larvae of flies resistant to DDT than to normal flies.—*U.S. Department of Agriculture.*

* * *

The First International Congress of Plastic Surgery, organized by the Scandinavian Association of Plastic Surgeons, will be held at Stockholm and Uppsala, Sweden, on 1-5 August 1955. Patron —H.M. the King of Sweden, and Honorary President—Sir Harold Gillies, London. The Headquarters of the Congress will be the Concert Hall (Konserthuset), Stockholm. The official language of the Congress will be English, though contributions may be presented in any language, and there will be no simultaneous interpretation service. The Congress is planned to cover the whole

field of plastic surgery; the papers preliminarily announced seem to focus special interest on the treatment of congenital deformities (cleft lips and palates; abnormalities of the external genital organs) and the surgery of the facial bones, particularly the mandible. Membership fee Swed. Kr. 75 and for accompanying family Swed. Kr. 30; this should be remitted before 1 June 1955 by draft on the Svenska Handelsbanken, Stockholm, to the account of Congress. All communications should be addressed to Dr. Tord Skoog, M.D., First International Congress of Plastic Surgery, Uppsala, Sweden.

* * *

New Microscope. A new electronic microscope now being made can enlarge red blood-cells so that they appear to be bigger than cricket balls. Living cells can be enlarged up to 15,000 times with the instrument, which can project them in full colour on to a 6-foot screen for teaching purposes or on to a television set for rapid consultation between a surgeon and pathologist while an operation is in progress.—*C.B.S. Laboratories.*

* * *

Cape Town Paediatric Sub-Group. The next meeting of this subgroup will be held on Friday, 3 June 1955 in the E Floor Lecture Theatre, Groote Schuur Hospital, at 8·15 p.m. This will be a joint meeting with the Physicians Group, the subject being, *Some Aspects of Congenital Heart Disease*. The speakers for the evening are Drs. L. Vogelpoel and M. Nellen.

* * *

Royal College of Physicians of Edinburgh. At a Quarterly meeting of the College on 3 May 1955, the President, Sir Stanley Davidson, in the Chair, the following *inter alia* were elected members of the College: L. Molk, M.B., Witwatersrand, S. S. Levin, M.B., Witwatersrand, E. S. Nash, M.B., Cape Town, M. A. Kibel, M.B., Witwatersrand.

POLIOMYELITIS IN THE UNION

Following are the returns, supplied by the Union Department of Health, of cases notified under the Public Health Act as suffering from Poliomyelitis in the period 29 April to 5 May and 6 to 11 May.

Report for the period 29 April to 5 May.

	European	*Non-European*
Transvaal:		
Benoni		1
Total for Transvaal		1
Cape Province:		
Cape Divisional Council	1	1
Matatiele District		1
Grabouw Municipality	1	
Total for Cape Province	2	2
TOTAL FOR THE UNION ..	**2**	**3**

Report for the period 6 to 11 May, 1955.

Cape Province:		
De Doorns Municipality	1	
Cape Divisional Council		1
Worcester Municipality	1	
Total for Cape Province ..	2	1
Orange Free State:		
Verkeerdevlei district Brandfort ..		1
Welkom Municipality	1	
Total for Orange Free State	1	1
Natal:		
Eshowe		1
Ixopo		1
Total for Natal	0	2
TOTAL FOR THE UNION ..	**3**	**4**

Union Department of Health Bulletin. Report for the 6 days ended 11 May 1955.

Plague, Smallpox, Typhus Fever: Nil.

Epidemic Diseases in Other Countries:

Plague: Saigon-Cholon (Viêt-Nam).

Cholera in Calcutta (India); Chalna, Dacca (Pakistan).

Smallpox in Moulmein, Rangoon (Burma), Phnom-Penh (Cambodia); Allahabad, Bombay, Jodhpur, Kanpur, Kozhikode, Lucknow, Madras, Tellicherry (India); Dacca, Karachi (Pakistan); Nhatrang (Viêt-Nam); Mogadiscio (Somalia); Tanga (Tanganyika).

BOOK REVIEWS : BOEKRESENSIES

NETHERLANDS CONTRIBUTIONS TO SOUTH AFRICAN MEDICINE

Contributions of the Netherlands to the Development of South African Medicine (1652-1902). By H. S. N. Menko, Arts. (Pp. 152 with illustrations). Amsterdam: H.A.U.M., L/a J. H. De Bussy. 1954.

Contents: Introduction. 1. Johan van Riebeek, (*a*) His youth and family and medical training; (*b*) his journey to and arrival at the Cape; (*c*) his work at the Cape; (*d*) description of the diseases of the Natives and their treatment in the settlement. 2. Some Netherlands medical men after Johan van Riebeek. 3. The medical care up to about 1800. 4. The medical care in the 19th century. 5. The medical care during the Great Trek. 6. The work of the Netherlands Ambulances during the Boer War. Summary. Samenvatting.

This slim volume purports to sketch the Hollanders' contribution to medicine in this country. It starts with Jan van Riebeek and ends with the Netherlands Ambulances operating with the Boer forces in the South African War. In between it discourses over the Cape Hospital of the Honourable Dutch East India Company and the Hottentots, and there is a chapter wedged in between the rest on the Great Trek as well.

So little has been written about the medical history of South Africa that it can perhaps be argued that any contribution to this field is to be welcomed; and certainly the author's style of writing flows easily. (Readers of the *Journal* will recollect Dr. Menko's interesting and scholarly articles on Jan van Riebeek in the Festival number in 1952.)

This readability is countered by two features, however, which make the book unacceptable to the purist. The first is hardly the fault of the author: the work has been ungrammatically translated and improperly proofed, and sentences have the irritating habit of running into one another without punctuation. Secondly—and from a purely historical angle—the work is very superficial, and Dr. Menko's interpretation of South African history should be read with extreme caution.

Small mistakes abound (e.g. the statements on page 99 that the British troops landed *in Saldanha Bay* in 1806, and that General Janssens surrendered *for want of stores*, are plainly incorrect), and proper names are often misspelt (e.g. the historian G. McCall Theal's name is rendered repeatedly 'Mac Call Theall'). One feels that Dr. Menko has not done justice to the references he has quoted, nor has he adequately covered the field he set himself.

As a story this little book makes pleasant reading (if one can overlook its irritating grammatical errors), but as a serious historical work it falls far short of the mark as measured by our standards.

E.H.B.

ON HEADACHE

Headache: Diagnosis and Treatment. By Robert E. Ryan, B.S., M.D., M.S. (in Otolaryngology), F.A.C.S. Pp. 338. £2 16s. 3d. St. Louis: The C.V. Mosby Company. 1954.

Contents: 1. Introduction. 2. The Physiologic Basis of Head Pain. 3. Objectives of all Headache Treatment. 4. Differential Diagnosis of Head Pain. 5. History Taking. 6. Examination of the Headache Patient. 7. Histamine Cephalalgia. 8. Migraine. 9. Abdominal Migraine. 10. Ophthalmic Migraine. 11. Tension Headache. 12. Generalized Vasodilating Headache. 13. Psychogenic Headache. 14. Nasal Sinusitis Headache. 15. Sluder's Syndrome Headache. 16. Myalgia of the Head. 17. Mixed Type of Headache. 18. Head Pain of Otological Origin. 19. Acute Meningitis. 20. Trigeminal Neuralgia. 21. Glossopharyngeal Neuralgia. 22. Temporal Arteritis. 23. Brain Tumor Headache. 24. Brain Abcess. 25. Subarachnoid Hemorrhage. 26. Posttraumatic Headache. 27. Subdural Hematoma. 28. Lateral Sinus Thrombosis. 29. Alcoholic Headache. 30. Hypoglycemic Headache. 31. Cardiovascular Renal Headache. 32. Constipation Headache. 33. Headaches due to Bone Disease. 34. Headaches due to Blood Abnormalities. 35. Gynecological Headache. 36. Headache due to Intoxications (Poisonings). 37. Headaches due to Cardiac Diseases. 38. Headache due to Diseases of Endocrine Glands. 39. Headache due to Cervical Pathology. 40. Headache due to Cerebral Pathology. 41. Headache due to Infectious Diseases of Bacterial Origin. 42. Headache due to Diseases of Virus Origin. 43. Headache due to Infectious Diseases of Rickettsial Origin. 44. Headache in Diseases of Mycotic Origin. 45. Headaches in Diseases of Protozoan Origin. 46. Various Other Conditions Associated with Headache. 47. Allergic Headache. 48. Oral Cavity Head Pain. 49. Muscle Tension Headache. 50. Migraine in Children. 51. Postspinal Puncture Headache. 52. Ophthalmological Head Pain. Index.

This is a systematic text-book on Headache by an otolaryngologist. Most of the conditions discussed here are commonly seen in the everyday practice of the average physician, internist, ophthalmologist, neurologist and general practitioner.

With each form of headache problem the symptomatology usually found in the average case is given, although the important fact is stressed that all may have their atypical forms.

I have also noticed, with great joy, how much the author stresses that all-important fact that one of the most important factors in solving a headache problem, as in solving practically any medical problem, is the taking of a good history. In these days of special investigations and mechanical aids to diagnosis this fact seems worth while emphasising.

In passing I would like to refer to the interesting combination of drugs which is prescribed in the treatment of classical migraine, e.g. ergot, caffeine, an atropine derivative and a barbiturate. Personally I have had astonishing success in the treatment of migraine by this method.

This volume will be of interest to every member of the medical profession.

A.B.

MEDICAL EDUCATION

First World Conference on Medical Education. Held under the Auspices of the World Medical Association. Pp. 804+xvi. 60s. London. New York. Toronto. Geoffrey Cumberlege, Oxford University Press.

Contents: Opening Adresses. Section A—Requirements for Entry into Medical Schools. Section B—Aims and Content of the Medical Curriculum. Section C—Techniques and Methods of Medical Education. Section D—Preventive and Social Medicine. Concluding Reports of Vice-Presidents and Rapporteurs. Index of Contributors. Subject Index.

During August, 1953, there came together in London over 600 men and women, representing 127 faculties of medicine and 62 countries, for a 6-day conference on medical education. This substantial volume is a record of their proceedings. 'Those of us who planned the programme of the Conference', writes its President (Sir Lionel Whitby), 'decided to make no attempt at curriculum-building; such an attempt would only have ended in failure. By choosing individuals to speak on selected themes linked together in the four sections of the Conference we aimed rather at stimulating thought and even controversy . . .'

The 4 opening addresses are followed by the 79 papers which were read to the 4 sections into which the Conference divided; and included with each paper, or group of papers, is a summary of the main points made by the principal participants in the resultant discussions. The concluding reports are by the 4 vice-presidents, one of whom chaired each section of the Conference, and the 4 rapporteurs who supported them.

The Conference did not adopt any formal resolutions or recommendations on any of the topics which it considered. Its *Proceedings* enshrine no dogma, but are simply 'an encyclopaedia of factual information and considered opinion about medical education in all quarters of the globe'. One cannot review an encyclopaedia, but perhaps one or two quotations will give an impression of its quality.

'Section A reacted against the idea that science is in some ways inferior to the humanities as an instrument of education.... Let us remember our own deficiencies, intellectual and moral, and be content if we see only one or two divine attributes in our prospective students'.

In Section B: 'An analysis of the views which have been expressed upon the sort of curriculum likely to succeed in the twin aims of education and vocational training: the growth of medical knowledge has led to the fragmentation of the old primary teaching subjects into numerous sciences....all speakers seem to be agreed that the time has come to call a halt; to place the whole before the part, and to reunite the disintegrated fragments—in a word, to make the curriculum comprehensible once again....Opinion is strongly in favour of emphasising that the aim of medicine is to study and treat the sick person and not only the disease.'

In Section C: 'All were agreed on the value of using out-patient clinics and even the home, in addition to the hospital ward, as places of instruction....Of the 21 medical schools in the United Kingdom, general practice schemes are organised in 9.'

In Section D: 'The main task of medical education in the future is to turn out good doctors who are also equipped to be leaders of the health team....Many of the major preventive and therapeutic activities of social medicine must be taken through community political action. Doctors are afraid of this, but they should regard it as a challenge.'

G.W.G.

YEAR BOOK OF GENERAL SURGERY

Year Book of General Surgery. By Evarts A. Graham, A.B., M.D. Pp. 500 with illustrations. $6.00. Chicago: Year Book Publishers, Inc.,

Contents: 1. Introduction. 2. General Considerations. 3. Technical Contributions. 4. Shock, Fluids and Electrolytes. 5. Nutrition. 6. Wounds and Wound Healing. 7. Antibiotics. 8. Neoplasms. 9. The Scalp. 10. Face and Buccal Cavity. 11. The Neck. 12. Thyroid and Parathyroid. 13. The Breast. 14. Lungs and Pleura. 15. The Thorax and Mediastinum. 16. The Heart. 17. Hypertension. 18. Peripheral Arteries and Aorta. 19. Peripheral Veins. 20. Lymphatic System. 21. Abdomen—General. 22. Liver and Spleen. 23. The Biliary Tract. 24. The Pancreas. 25. The Esophagus. 26. The Stomach and Duodenum. 27. The Small Intestine. 28. The Colon & Rectum. 29. The Anus. 30. Hernia. 31. The Adrenal Glands. 32. The Genitourinary System. 33. The Extremities. Section on Anesthesia. Index.

A book edited by Evarts A. Graham, abstracting from original articles of the world's surgical literature, promises good stimulating reading, and this is certainly achieved in this 1954—1955 Year Book on General Surgery.

The introduction by the editor may cause a slight rise in the blood pressure of the reader outside the American continent, but gives food for thought and will serve to shake the reader out of any complacency which western European surgeons or their pupils may tend to fall into.

For the general practitioner as well as the specialist surgeon this book will be of interest. Because it presupposes a complete understanding of the subject, the undergraduate may be unable to view information gained from this book in its proper perspective; to supplement his basic reading however, and to give him an idea of the newer developments and trends in surgical research, it is worth reading.

One is really at a loss which sections to comment on. Each page contains some interesting facts; some remind one of an article previously read, others stimulate one to read the original article on the subject referred to. References are clearly indicated at the bottom of each page.

The editor's occasional cryptic note at the end of some of the sections gives one an insight into the man Evarts Graham; e.g. 'Everybody knows these things but they are sometimes forgotten' (p. 31).

Throughout the book the simple clinical test to demonstrate a lesion is stressed; e.g. Tourniquet Paralysis Syndrome (p. 390), or Diagnosis of Depth of Burning in the section on Wounds and Wound Healing (p. 45).

British-orientated surgeons will probably find an occasional statement which they would be less dogmatic about. In the section

on Thrombophlebitis and Phlebothrombosis there is no article on the use of anticoagulants in treatment.

The section dealing with Stomach and Duodenum demonstrates beautifully the differences in opinion held throughout the world on the best form of surgical treatment for peptic ulcer and its complications. The description of Gastrectomy with Replacement by F. Austin Henley in the Ann. Roy. Coll. Surg. Engl., September 1953, demonstrates that the search is still going on for the ideal method of surgical treatment.

Doubt is again cast on the long accepted congenital theory of the etiology of Pilonidal Sinus (p. 365). The editor's note: 'This is an interesting idea. Perhaps it is correct' will probably be altered to: 'It has been proved to be correct' in some future edition.

A very interesting section on Anaesthesia is included, edited by Stuart C. Cullen. In the space of 81 pages many points relating to the well-being of the surgical patient are discussed. This section discusses briefly the newer concepts of the physiology of circulation and ventillation. It touches on some of the problems and dangers encountered in the use of hypothermia and hypotensive, muscle-relaxant and other commonly used drugs. Work on premedication in children is included. The special risk attached to patients previously treated with cortisone, and who suffer from resultant adrenal cortical depression become shocked during operation, is mentioned.

Bleeding from the skin and subcutaneous tissues during cyclopropane anaesthesia (p. 428) gives an acceptable explanation for the observation, long since made by surgeons, of increased oozing from wounds when this anaesthetic is used.

The editor has succeeded in sifting the corn from the chaff of world surgical literature and presents it in so concise a form that, to extract from it its full value, this book will have to be read and re-read.

Printed on good paper in clear type and with a pleasing cover this book will be an often-used and pleasant addition to the surgeon's library.

J.H.H.

CORRESPONDENCE : BRIEWERUBRIEK

REFRESHER COURSES FOR G.P.S

To the Editor: In reply to the letter in the *Journal* of 23 April 1955 by Dr. T. B. de Bruyn I am instructed by my executive to bring the following information to his notice:

The Medical Graduates Association of the University of the Witwatersrand, Johannesburg has run courses in Paediatrics, Haematology, Cardiology, Ear, Nose and Throat and Skins and Medicine. These were started 3 years ago.

In conjunction with the Department of Surgery of the University of the Witwatersrand, our Association will hold an intensive week-end course in Surgery at the end of July, and it is hoped to hold a similar course in Gynaecology later in the year.

These courses have been very popular and successful. Members have attended from as far afield as Ermelo, Welkom, Stanger, Fort Beaufort, Stutterheim, Pietersburg and Klerksdorp.

Although they are primarily for members of the Medical Graduate Association of the University of the Witwatersrand, applications from graduates of other universities are considered, and if at all possible, accepted.

R. Burns,
Hon. Secretary

Medical Graduates Association
University of the Witwatersrand
Medical School
Hospital Street
Johannesburg
9 May 1955

ABUSE OF OFFICIAL TRANSPORT

To the Editor: I have to advise you that the attention of this Board has again been drawn to the continued abuse of the ambulance services, in that medical practitioners are ordering official transport when they could use either public or private transport for their patients.

The number of ambulances in use is not sufficient to cope with the increasing demand and this naturally leads to unnecessary delays and consequent dissatisfaction on the part of patients definitely requiring ambulance transport.

It would be appreciated if your Journal will give this matter the publicity it deserves and request medical practitioners to co-operate by ordering ambulances only when absolutely necessary.

H. M. Timoney
Chairman

Teaching Hospitals Board (Cape Town)
6th Floor, Industry Building
58 Loop Street
Cape Town
16 May 1955.

SPECIALIST REGISTRATION: PRACTITIONERS WHO HAVE BEEN ON MILITARY SERVICE

To the Editor: At its meeting in September 1954, the Specialists Committee of the Council passed the following resolution:

'That it be recommended to Council that its instruction relating to practitioners who have been on military service for two years prior to July 1945, be withdrawn as from 1 January 1957'.

The instruction of the Council referred to above, provides for the consideration under the provisions of rule 12 of the rules for the registration of specialities of applications by practitioners who have been on military service for at least two years prior to July 1945. The instruction which has been operative since March 1948, reads as follows:—

'That it was the intention of the Council that in the application of rule 12 to ex-service medical officers each case should be judged on its merits but that the standard demanded should be approximately that of the rules in force prior to January 1948.'

The recommendation of the Committee was considered and adopted by the full Council at its meeting in March 1955, and I have been instructed by the Council to request you kindly to publish in your Journal the fact that the above instruction of Council will be withdrawn as from 1 January 1957.

W. H. Barnard
Assistant Registrar

S.A. Medical and Dental Council
P.O. Box 205
Pretoria

ANAL FISSURE

To the Editor: In the *Journal* of 30 April under 'Revision Series', an article[1] on *The Surgery of Ano-Rectal Conditions including Haemorrhoids* from the Department of Surgery, University of the Witwatersrand, discusses briefly the surgery of anal fissure. It states that the only sure method for the cure of the chronic anal fissure is the adequate excision of the fissure and sentinel pile and sectioning of the subcutaneous sphincter.

I wish to state, categorically, that the subcutaneous external sphincter plays no part either in the cause or cure of a chronic fissure. The cure of the chronic fissure is carried out by linear division of the internal anal sphincter as described by me in 1951, 1953 and 1954 in articles [2], [3], [4] in the *Journal* on ano-rectal surgery. This method is now exclusively practised and taught by all the surgical staff of St. Mark's Hospital for Rectal Diseases, London. At Johannesburg, also, a large number of these cases are now dealt with, on the ambulatory system, in the out-patient department.

Stephen Eisenhammer, M.B. (Ed.), F.R.C.S. (Eng.)

34 Moray House
Cr. Jeppe and Smal Streets
Johannesburg
16 May 1955

1. Skapinker, S. (1955): S. Afr. Med. J., **29**, 407.
2. Eisenhammer, S. (1951): *Ibid.*, **25**, 486.
3. *Idem.* (1953): *Ibid.*, **27**, 266.
4. *Idem.* (1954): *Ibid.*, **28**, 264.